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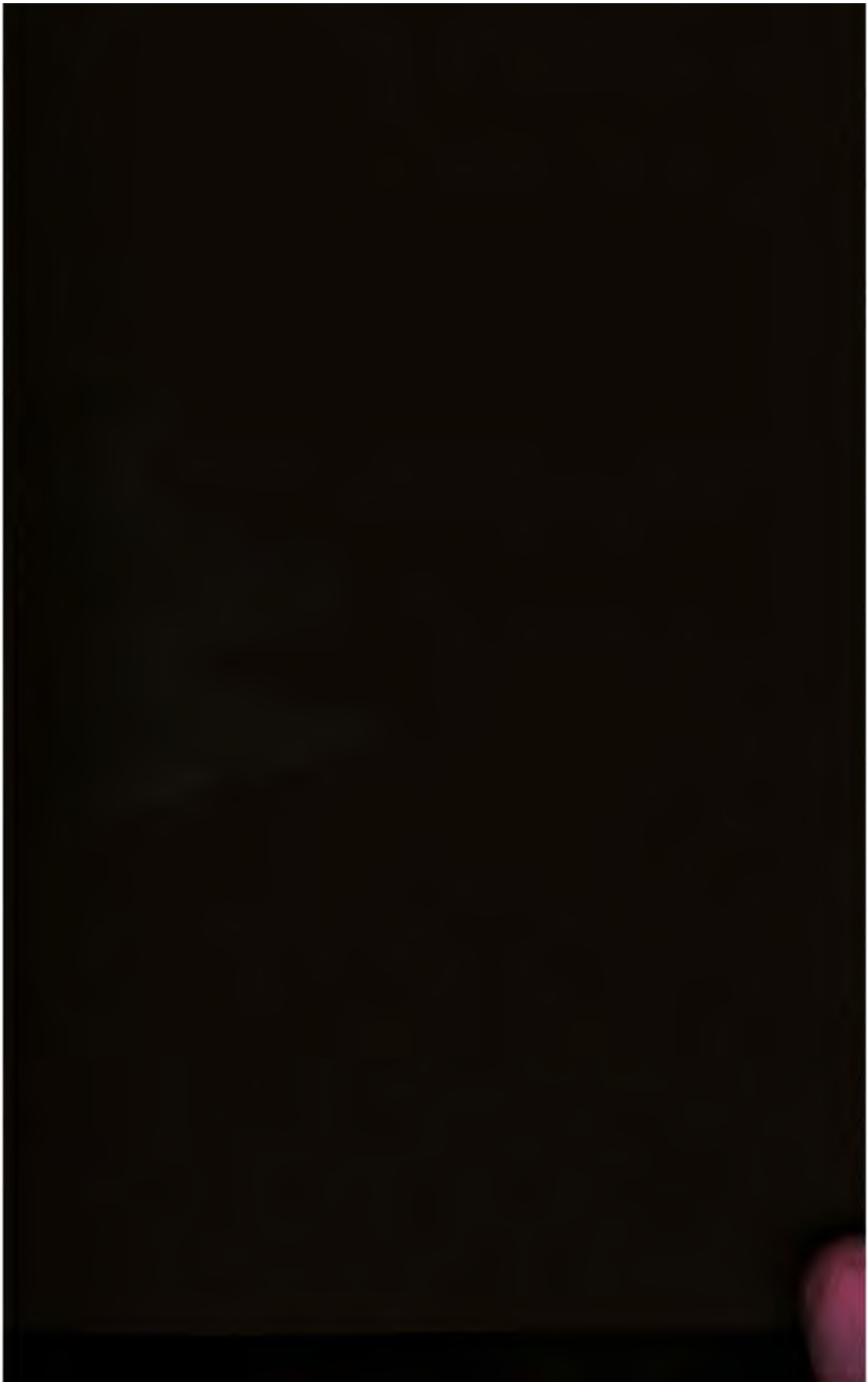

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

THIRD SESSION.

HELD IN WASHINGTON, D. C., SEPTEMBER 22 TO 25, 1891.

EDITED BY

WM. PERRY WATSON, A.M., M.D.,

SECRETARY.

VOLUME III.

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WILLIAM P. NORTHRUP, M.D.....New York.
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LOVE, I. N., M.D. ..... St. Louis.
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MINUTES

OF THE

THIRD ANNUAL MEETING

OF THE

AMERICAN PEDIATRIC SOCIETY,

HELD IN WASHINGTON, D. C., SEPTEMBER 22 TO 25, 1891.

September 22, 1891.—*First Day, Evening Session.*

THE President, T. M. Rotch, M.D., of Boston, called the meeting to order at 8 o'clock, in parlors 182 and 183 of the "Arlington."

The following members were present, besides a large number of visiting physicians:

S. S. Adams, Washington; W. D. Booker, Baltimore; S. C. Busey, Washington; W. L. Carr, New York; F. Forcheimer, Cincinnati; J. H. Fruitnight, L. Emmett Holt, A. Jacobi, Henry Koplik, New York; T. S. Latimer, Baltimore; W. P. Northrup, Joseph O'Dwyer, New York; William Osler, Baltimore; C. P. Putnam, T. M. Rotch, Boston; A. Seibert, J. Lewis Smith, New York; V. C. Vaughan, Ann Arbor; William Perry Watson, Jersey City.

On motion, the minutes of the last annual meeting were approved as published.

On motion, it was ordered that all the papers presented to this meeting, with the discussions thereon, be furnished ex-

clusively to the ARCHIVES OF PEDIATRICS for publication therein; the publishers in turn agreeing to make three hundred reprints thereof for the Society free of all expense, and similar to those furnished at the last meeting.

The Secretary read a letter from Dr. William Pepper, Chairman of the Executive Committee of the Congress, saying that the American Pediatric Society had been duly admitted to participation in the Congress of American Physicians and Surgeons.

Letters regretting inability to be present were read from Drs. Blackader, Escherich, Earle, Edwards, Huber, Keating, Love, Starr, Waxham, and Wilson.

The President, Dr. T. M. Rotch, then delivered his annual address.

Dr. J. Lewis Smith read a paper, "How to Prevent Complications and Sequelæ in Scarlet Fever."

The subject for discussion, arranged by the Council, viz., "The Diagnosis of Pneumonia in Infancy and Early Childhood," was then taken up.

A paper by Dr. T. S. Latimer on (1) "The Most Diagnostic Symptoms of the Early Stage of Lobar Pneumonia and the Differential Diagnosis from such Diseases as Meningitis, Malaria, Scarlet Fever, etc.," was read by title, in the unavoidable absence of the author. (2) "The Diagnosis of Consolidation of the Lung from Effusion (Serous or Purulent) and the Differential Diagnosis between Lobar Pneumonia and Broncho-Pneumonia," by Dr. F. Forchheimer. (3) "The Diagnosis of Broncho-Pneumonia (Acute and Chronic) from Tuberculosis," by Dr. William Osler. (4) "The Diagnosis of Broncho-Pneumonia from Bronchitis; also the Temperature Range in Acute Pneumonia, both Broncho and Lobar," by Dr. L. Emmett Holt.

The general discussion was participated in by Drs. Koplik, Jacobi, Smith, Holt, Osler, Northrup, the President, and Adams.

On motion, the Society adjourned at 11 P.M.

Second Day.—Morning Session.

The Society, after a very enjoyable breakfast with the President, was called to order at 11 o'clock by the President.

Dr. William Travers Howard, Jr., Baltimore, read a paper (by invitation) on "A Case of Congenital Heart-Disease," and exhibited the heart.

Dr. Seibert read a paper entitled "Further Report on Submembranous Local Treatment in Pharyngeal Diphtheria," which was discussed by Drs. Koplik, Northrup, the President, Smith, Seibert, Holt, Fruitnight, and Putnam.

Dr. W. D. Booker read a paper entitled "A Case of Ulcerative Catarrhal Dysentery."

On motion, the Society adjourned at 1.30 P.M.

Third Day.—Morning Session.

The Society was called to order by the President at 9 o'clock.

The Recorder read a paper entitled "Tubercular Osteitis of the Hip, originating in Colorado," by Dr. J. M. Keating.

A paper entitled "One Hundred and Forty Cases of Heart-Disease in Children," by Dr. F. M. Crandall, New York, was read by title.

A paper entitled "Manifestations of 'La Grippe' in Children," by Dr. Charles Warrington Earle, was read in the abstract by Dr. Seibert.

Dr. Joseph O'Dwyer read a paper entitled "Exhibition of Laryngeal Tubes for the Performance of Forcible Respiration," with demonstration of instruments, which was discussed by Drs. Northrup, Putnam, and O'Dwyer.

Dr. William Osler read a paper entitled "The Association of Congenital Wry-Neck and Facial Herniatrophy."

Dr. Charles G. Kerley read (by invitation) a paper entitled "The Application of Gavage in the Treatment of Uncontrollable Vomiting in Infants," which was discussed by Drs. Holt, O'Dwyer, Putnam, Koplik, Seibert, and Kerley.

Dr. Northrup read a paper entitled "Scorbutus in Children," which was discussed by Drs. Rotch, Northrup, Holt, Fruitnight, Putnam, Jacobi, and Smith.

Dr. Adams exhibited a case of pseudo-hypertrophic paralysis.

Dr. Holt read a paper entitled "Two Cases of Acute Primary Nephritis in Infants."

A paper by Dr. Jackson, entitled "Chronic Nephritis in Children," was read by Dr. Putnam.

Dr. Smith read a paper entitled "Scarlatinal Nephritis in Children."

The President announced that the last three papers were opened to discussion, which was participated in by Drs. Seibert, Forchheimer, Putnam, Koplik, Holt, Smith, Fruitnight, Northrup, Rotch, Jacobi, and Carr.

On motion, the Society adjourned at 1.30 P.M.

Third Day.—Evening Session.

After a banquet in the "Arlington," the Society was called to order by the President at 10 o'clock, to receive the report of the Council. The Council reported the following nominations to offices and membership, and, on motion, it was resolved that the report be accepted and the nominations to offices confirmed.

President.—William Osler, M.D., Baltimore.

First Vice-President.—J. M. Keating, M.D., Colorado Springs.

Second Vice-President.—F. Forchheimer, M.D., Cincinnati.

Secretary.—S. S. Adams, M.D., Washington.

Recorder.—William Perry Watson M.D., Jersey City.

Treasurer.—C. W. Townsend, M.D., Boston.

For member of Council, W. D. Booker, M.D., Baltimore.

As representative on the Executive Committee of the Congress, Dr. A. Jacobi; as alternate, Dr. T. M. Rotch.

The following new members were elected by ballot: E. M. Buckingham, M.D., Boston; F. M. Crandall, L. M. Yale, New York; B. K. Rachford, M.D., Newport, Kentucky; I. H. Snow, Buffalo.

The names of Drs. E. C. Wendt, New York, and H. C. Haven, Boston, were dropped from the roll of membership, as per the Constitution.

An assessment of five dollars on each member was ordered.

It was also recommended and ordered that the Society should meet in Boston in the last week in May, 1892.

The following amendments to the Constitution were also recommended by the Council, and, according to the Constitution, will be acted upon at the next annual meeting:

To Article III.: "The membership shall be limited to sixty, and that every applicant for membership shall accompany his application by a thesis of his own composition, and printed copies or references thereto of papers published by him and of the official positions held by him, as well as such other scientific work in Pediatrics as he may have accomplished; and, furthermore, that membership shall be completed by the signifying of acceptance of election and a payment of an initiation fee of ten dollars."

To Article XII.: "Or for non-payment of dues for one year, two notifications being given."

Fourth Day.—Morning Session.

The Society was called to order at 9 o'clock by the President.

Dr. Forchheimer read a paper entitled "Etiology of Stomatitis Aphthosa," which was discussed by Drs. Koplik, Putnam, Forchheimer, and the President.

On motion, it was ordered that Drs. Forchheimer and Rotch be constituted a committee to revise and report to this Society, at its next annual meeting, "A Nomenclature of Stomatitis."

Dr. Fruitnight then read a paper entitled "Perityphlitis in the Young," which was discussed by the President and Dr. Putnam.

On motion, a vote of thanks was unanimously tendered to President Rotch for the careful and painstaking way in which he had administered the duties of his office during the past year.

On motion, the Society adjourned to meet in Boston in 1892.

WILLIAM PERRY WATSON,
Recorder.

THE PRESIDENT'S ADDRESS.

ICONOCLASM AND ORIGINAL THOUGHT IN THE STUDY OF PEDIATRICS.

BY T. M. ROTCH, M.D.,
Boston.

GENTLEMEN,—Fellow-members of the American Pediatric Society, three years have passed since, in this very city, we crystallized into being and became an entity. Again we gather together to emphasize our individuality as a recognized society. Now, however, as a part of a national organization. Ourselves national in every sense of the word, with our members enrolled from the shores of the Pacific, from the South, from what was formerly the West now the great centre of our commonwealth, from the Atlantic coast, and then stretching still farther to the North, from our sister country, Canada.

I bid you welcome to our national Capital! I congratulate you on the progress which every year is being made in the study of a most interesting as well as important branch of medical science. In the branch which is to help make the people of the future in our great republic worthy of the responsibilities which such a republic brings with it. Which is to make our American citizens both physically and mentally, year by year, decade by decade, superior to their forefathers, and thus enable them to develop their country and its vast resources; thus make her stand shoulder to shoulder, neck to neck, in the race for supremacy which is going on all the world over. A young country in comparison with our medical competitors in the old world, well fitting is it that our Society should devote itself to a subject essentially young not only in years, but young in the knowledge which up to the present time has been acquired concerning it.

So much less has been done in the proper and scientific study of children than at other periods of life, that it is no wonder that we have entered upon the especial investigation of and research in this branch of anthropology with the keen interest of explorers in an almost unknown country. Of still further interest, also, when we discover not only that there is a

vast expanse of unknown, but that much which was supposed to be known is in reality a poor subterfuge of unreal facts forming structures of misleading results, which in the scientific medicine of adults would not for a second be tolerated; in fact would be laughed to scorn as relics of the dark ages of necromancy. This same misnamed medical knowledge, however, when representing the infant and child, has been accepted with but little question. What our society was needed for, what it was formed for, what it intends to do, is to place the study of pediatrics on the same elevated plane that has been established for adult life.

To accomplish this, it must in the early years of its existence be iconoclastic.

It must break down and sweep away these misleading structures, clear the ground of these undesirable remnants of the past, get down to the virgin soil, and then, by original research, build up our new fabric on a stable basis.

Wise iconoclasm and patient originality must be the weapons by which we shall fight our way to the front and place the standard of pediatrics where it ought to be,—place it side by side with the already perfected anatomical and physiological investigations which have become the true basis for the enlightened clinical study of human beings.

To intelligently understand the fully-developed man in health and disease, it seems self-evident that the anatomy and physiology not only of the final state of growth should be studied, but also that the various stages of development, from embryo to infant and infant to child and child to adult, should successively be dealt with. This in the past, however, has been but little done. On the contrary, the very opposite method has been adopted; the most careful attention being paid to adult anatomy and physiology, and then deductions made backward from adult to child,—a retrograde means of acquiring knowledge, which has proved eminently unsuccessful.

In the Old World, as well as in the New, we find that these false methods have been pursued. What little has by more rational methods of study been accomplished in the investigation of infants and children has as large a place in America as in Europe. Here, then, is our opportunity for original

research, for we have a branch of medicine which universally is new. We have the same advantages in clinical material, in well-equipped laboratories, in special hospitals, as are to be found anywhere. Let us be sure that we give as much, if not more, to the savants of Europe as we receive from them.

As I look upon the members of our Pediatric Society and see how well fitted they are to be leaders in the several communities wherein their lot is cast, it is impressed upon me that, when meeting as a whole, our Society should represent advanced and general ideas; should deal with living questions of the day, and that the results of our deliberations should authoritatively be promulgated by our individual representatives far and wide throughout our whole country.

Thus only can the unenlightened influence of the profession at large on the laity be curbed in the harm it is continually doing to scientific medicine; thus only can the self-sufficient ignorance regarding the most critical period of human existence be properly combated, and the general physician be forced to understand that he has but a limited knowledge of what he has been in the habit of considering simple questions; of what he has been accustomed to give off-hand opinions and advice on to the credulous mother of the suffering child.

You probably all have met with the same experience as mine, not only among the poorer class of physicians, but, astonishing as it still seems to me, among the highly educated and distinguished members of our profession.

Among men who are recognized leaders; men who have done much for humanity in other branches of medicine, and yet who, with dignified authority, continue to utter dead platitudes concerning children; platitudes which have been handed down from their forefathers or copied from the already cumbersome literature of pediatrics, and again enunciated by themselves in their own writings.

It is no exaggeration to state that a large number of sick infants and young children throughout the land are suffering from the vigorous treatment of their zealous medical attendants, rather than from the disease with which they started. This should be stopped. This it is the mission of our Society to put an end to.

I ask you to carefully consider this vital question. I ask you to attack it with iconoclasm and original thought, for, I assure you, it will repay you by opening up vast fields of intensely interesting brain-work.

It is not difficult to pick out instance after instance of the truth of what I have just said.

The therapeutics of infancy and childhood, as understood by thousands of practitioners in Germany and France, in England and in America, is a wonderful exhibition of what vagaries the human mind, sound on other subjects, can be induced to indulge in. A total disregard of natural processes; an over-exaggeration of symptoms, which in the adult mean danger and require active treatment, while in the child they are but simple and harmless manifestations of an over-excited nervous system; such is the *Ignis Fatuus*, so misleading not only to those who have not especially studied the early stages of life, but also to those who have seen much of it, but with eyes blinded by traditions of the past. It would seem as though in this age of rapid discovery, of quick change and interchange of ideas,—in an age when the precision of our investigators in all branches of research has almost placed the much-abused profession of medicine on a level with the exact sciences,—it would seem, I say, that we, who are endeavoring to place our especial branch of study on firmer ground, should consider with unusual care the opinions which we express to the profession at large and to the laity; that our writings should be founded on principles as accurate as the science of the day can make them.

This, however, in the past has been but little done, and consequently we have amassed volumes of most unreliable pediatric literature.

I honestly believe that there is reason for saying this.

I truly think that it is one of the vital living questions of the day which we should appreciate and grapple with.

What we need to aid us in this reform—for a reform it must be in every sense of the word—is a fitting humility as to the degree of our knowledge; a proper sense of how small has been the true working capital which we have had at our command in the study of pediatrics.

We must confess, and by so doing we certainly add new strength to our writings, that the medical science of the present day is too vast for one mind to master as a whole; for one man to attempt to be an expert in.

We must make use of the experts in their several branches, whether it be chemistry or physiology, anatomy or bacteriology, to strengthen and make stable our general deductions as clinical investigators.

In this way only can we, in an age essentially one of progress in details, prepare ourselves to produce work of true original thought, of real intrinsic value.

Work which, with its lustre of matured and well-proved thought, spreading far into the future, will be deemed worthy to be placed with the other records of a lasting literature.

The literature of a society gives evidence of the work which has been done by its members long after their individual labors have ceased, and their places have been filled by a younger generation.

If that literature is well considered, and shows evidence of representing not the hasty judgment of the day but of being built on the broad foundation of exact science, it will be worthy of the study of our descendants.

As a sound unquestioned nucleus it will give encouragement to broaden the field of inquiry and to add fresh knowledge year by year.

Let us then see that the circumference of our literary circle, small as it now is, shall spread wider and wider, honestly fitting itself for the far-outstretching, eternal criticism which will judge our work long ages after we are dead and gone.

Let us leave to the coming ages a fitting centre for that scientific, human, eternal progress spoken of in the old hymn which says,—

"Eternity! Eternity!
How long art thou, eternity!
A ring whose orbit still extends,
And ne'er beginning, never ends,
Always thy centre, ring immense,
And *never* thy circumference,
Mark well, oh, man, Eternity!"

It should be our aim that a lasting literature of this kind, ever young and fresh in the truths which it enunciates, ever presenting a solid centre for more extended investigations, should emanate from members of our Society.

Again, then, let us see that what we write, that what we publish, not only in our transactions but in our books, and in the medical journals of the day, shall form volumes worthy of the admiration and not of the derision of our successors.

Let our books so teem with unquestioned truths, with pages recording facts, and finding no place for theories, that on their face they will prove the truth and not the fallacy of the lines which, in the present age of unbridled literature, might well be questioned. Lines which read,—

> "Give books: they live when you are dead;
> Light on the darkened mind they shed:
> Good seed they sow, from age to age,
> Through all this mortal pilgrimage.
> They nurse the germ of holy trust;
> They wake untired when you are dust."

THE DIAGNOSIS OF PNEUMONIA IN INFANCY AND EARLY CHILDHOOD.

I.—THE MOST DIAGNOSTIC SYMPTOMS AND SIGNS OF LOBAR PNEUMONIA (EARLY STAGE), AND THE DIAGNOSIS FROM SUCH DISEASES AS MENINGITIS, MALARIA, SCARLET FEVER, ETC.

BY THOMAS S. LATIMER, M.D.,
Baltimore, Md.

By lobar pneumonia is here meant croupous pneumonia or pneumonic fever, characterized by an inflammatory exudate in which is a relative preponderance of fibrin. An infectious, not contagious, self-limited disease (Minot). The tendency of modern opinion is to consider it a constitutional affection, having its origin in a specific germ, probably the capsule-coccus of Friedlander, with a local pulmonary manifestation. The evidence is, however, as yet far from conclusive; the

absence of this organism in many cases, its presence in the mouth of healthy subjects, the readiness with which pneumonia appears to be induced by the injection of various other morbid materials, and the claim preferred by different observers for the causal relation of different micro-organisms, make it prudent to consider this question as yet unsettled. Moreover, it is seldom that this disease is communicated from one person to another. My own experience in thirty years would prompt me to say it is never so communicated. Whilst it is also doubtless true that in some cases the febrile action sustains no direct relation to the local affection, yet I think it cannot be questioned that usually it does, and that some of the most characteristic symptoms, including pyrexia, are directly as the extent and severity of the pulmonary involvement. A sudden rise of fever in the stage of defervesence is usually held—correctly, I believe—to indicate an extension of the local lesion. However much we may desire to refer lobar pneumonia to the class of affections whose origin is determined, the evidence as yet presented does not warrant any such positive conclusion.

Lobar pneumonia in childhood is usually ushered in by a well-marked rigor, speedily followed by rapid elevation of temperature; in infancy and early childhood frequently ranging from 103° F. to 106° F., with morning subsidence and evening rise of one or more degrees, and a sudden and generally permanent depression about the seventh to ninth day. This temperature range is, perhaps, the most characteristic feature of the disease. It is almost invariably associated with cough, which in early childhood is not accompanied by expectoration, so that one of the most distinctive signs in the adult (the rusty sputum) is lacking at this period. Pain is, as in the adult, commonly present, and is increased in coughing and in prolonged inspiration; but it is not so commonly referred to a definite point, as, in the adult, to region of the nipple of the affected side. When the child is old enough to locate it, it is frequently referred to the epigastric region. The pain, doubtless, indicates involvement of the pleura; though in cases where the cough is very frequent and labored, muscular pain—diaphragmatic or intercostal—may be induced. The patient usually prefers to lie on the affected side or back.

The pulse is frequent, ranging from 120 to 160 per minute, soft and full at the outset, but soon becoming small and irregular (Loomis). Its ratio to respiration may fall as low as two and three-tenths to one (Minot).

Respiration is always quickened disproportionately to the pulse, and is sometimes labored. When unaccompanied by bronchial involvement, however, the respiration, though frequent, is not usually difficult. Although, as in pyrexia, under other conditions respiration may be greatly disturbed independently of the pulmonary lesion, yet the disturbance nevertheless is commonly closely associated with it.

Among the earliest symptoms of lobar pneumonia is vomiting, preceded by a feeling of general malaise and accompanied by headache and pain in other regions of the body. There is little or no appetite. This gastric disturbance is probably of nervous origin, and is closely associated with that nervous perturbation that frequently issues in convulsions. This is probably due to the close physiological relations existing between the brain and stomach in early childhood. Except in old alcoholic subjects, these nervous phenomena are not common to this affection in adults.

A well-marked flush on one or both cheeks is so commonly present and exceptional in other febrile conditions, except phthisis, that it is a valuable diagnostic sign. The cheek on which the flush appears, when it is limited to one side, has no constant or necessary relation to the side on which the pneumonitis is.

It appears that the apex is far more liable in children than in adults. The crepitant râle is less constantly present or more difficult to determine, and is preceded or substituted by gradually diminishing respiratory murmur. The chlorides in the urine are absent or greatly diminished.

All the symptoms and signs enumerated—chill, fever, cough, pain, frequent pulse, respiration disproportionately rapid, fine crepitation, flushed cheeks, dulness on percussion, diminishing respiratory sounds, bronchial breathing, with headache and other nervous phenomena—may all be present in the first stage, the stage of engorgement. Why, then, with so marked a clinical history, should there be any difficulty in diagnosis, even in the first stage?

The difficulty is due to the fact that in no case are all these symptoms present; very frequently the patient is not seen until this stage is passed; no one of the symptoms or signs is absolutely pathognomonic except the crepitation, which cannot always be heard, even with well-behaved children; the rusty sputum, so distinctive in the adult, is seldom to be seen in young children; cases are recorded in which auscultation and percussion revealed nothing until the fourth or fifth day.

The supposed specific germ is not readily obtained from children, and is not diagnostic when found.

Yet, with care, even in the first stage, lobar pneumonia may generally be recognized by a competent observer. Fever is always present, and usually presents the characteristic curve. The pulse is always rapid. The respiration invariably disproportionately quick. The consequent indisposition to cry or violently resist examination is distinctive. Bronchial breathing may very generally be heard even when crepitation is not. Lagging of the affected side is almost invariable.

The diseases with which it is most frequently confused are bronchial or catarrhal pneumonia, capillary bronchitis, tuberculosis, pleurisy, meningitis, and, enumerated among those from which it is to be differentiated in the title of this paper as presented to me, are scarlet fever and malaria.

From bronchial pneumonia it may be distinguished in the first stage by the following symptoms and signs:

Lobar Pneumonia.	*Bronchial Pneumonia.*
Occurs abruptly.	Is often slowly developed.
Primary.	*Usually secondary.*
Commonly unilateral, and limited to one or two lobes. If primary and at the apex, it can be pronounced.	Commonly bilateral. Has no definite limitation; does not usually involve an entire lobe.
Cough frequent, but not constant, and does not usually precede pyrexia.	Cough invariably present; generally precedes pyrexia.
Characteristic temperature curve. Unilateral elevation.	Temperature irregular and equal on both sides.
Respiration disproportionately quick, but not labored.	Respiration disproportionately quick and labored.
Pulse rapid, but soft and full.	Pulse rapid, and often weak and unresisting.

Fine crepitation limited to circumscribed region, usually on one side only.	Coarse moist râles, bilateral and diffused.
Limited and defined dulness on percussion, and	Dulness and fremitus, when present, more diffused.
Vocal fremitus and resonance.	Vesiculo-tympanitic sound due to emphysematous lobules.
Nervous symptoms more distinctive and severe.	Nervous symptoms less frequent and less severe.
Flushed cheek.	Lividity of face.
Pain severe.	Pain less severe.

From capillary bronchitis it is differentiated by the following symptoms:

Lobar Pneumonia.	*Capillary Bronchitis.*
Onset abrupt, with rapid elevation of temperature; higher on affected side.	Onset less sudden, temperature equal on both sides, and lower.
Respiration rapid, but not labored. No cyanosis.	Respiration more rapid, very labored, with cyanosis.
Fine crepitation, unilateral and localized.	Subcrepitation, bilateral and diffused.
Bronchial breathing.	No bronchial breathing.
Slight localized dulness and fremitus.	No dulness in first stage, nor fremitus; exaggerated resonance above, diminished resonance in lower collapsed or œdematous lobes.
Cough sometimes absent; usually of moderate intensity.	Cough invariably present, almost continuous, and frequently attended with cyanosis.
Flushed cheeks.	Lividity of face.
Pain in side often severe.	Pain seldom present.

From acute inflammatory tuberculosis it is distinguished by—

Lobar Pneumonia.	*Acute Inflammatory Tuberculosis.*
Abrupt invasion, high persistent fever, usually preceded by chill.	Family history, usually slower development; fever often high, but irregular; repeated chills.
Frequently occurs under five years.	Comparatively infrequent under five years.
Pneumococcus. Hectic flush, expectoration of mixed blood (rusty sputum), no shreds of elastic tissue, no night sweats.	Tubercle bacillus. Hectic flush, expectoration, when patient is old enough, of unmixed blood; shreds of elastic tissue, night sweats.

Fine crepitation, usually limited to one lobe, with similar limitation of dulness and fremitus, and bronchial breathing.	Coarser crepitation, not so definitely limited, with gradually extending incomplete dulness from more than one point; less pronounced, but more extended, bronchial breathing and fremitus; bilateral.
But little emaciation.	Rapid and continuous emaciation.
Other organs seldom involved.	Other organs commonly involved.

The diagnosis of acute tuberculosis is well summed up by Dr. Stokes, of Dublin, in the following statement: "If, in a case presenting the symptoms and signs of intense bronchitis, or if crepitating râle has been present, yet persisting to the last, we find the chest becoming dull; if this dulness be extensive, yet incomplete, without bronchial respiration, the stethoscope showing that the lung is everywhere permeable, the solidity only occurring in points; or, if the crepitus be so slight as not to account for the dulness, we may make the diagnosis of the acute inflammatory development of tubercle." (Stokes, Sydenham, 1882.) If to this is added the detection of the tubercle bacillus, the evidence is complete.

Lobar Pneumonia.	*Pleurisy.*
High temperature, 103°.	Low temperature, 100°.
Rapid breathing without inspiratory catch, unless associated with pleurisy.	Rapid breathing, with inspiratory catch.
Pain not marked, except when associated with pleurisy.	Pain acute and uniformly present.
Cough loose; characteristic sputa when patient is old enough to expectorate. Pneumococcus.	Cough dry and hacking; expectoration not characteristic.
Fine crepitation, with slight dulness, frequently at the apex; vocal resonance and fremitus increased.	Friction sound often wanting in children, but when present, usually central. Dulness usually at the base; vocal resonance and fremitus absent over area of dulness. Varying site of dulness with changed position frequently occurs. Cardiac displacement. Fluid may be withdrawn with hypoderm.

Acute meningitis may be distinguished by its relative infrequency, the rarity with which it occurs apart from aural and

mastoid complications; by the absence of severe initial chill and subsequent high temperature; by slowing and irregularity of respiration and pulse, and by the absence of abnormal respiratory and percussion sounds, and by the absence of pain in chest or epigastrium.

The nervous phenomena are much the same in meningitis and in those cases of lobar pneumonia that have marked cerebral symptoms; but even those pneumonic cases where the cerebral symptoms precede the auscultatory and percussion phenomena are sufficiently distinguished by the high temperature, rapid breathing, and quick pulse.

With scarlet fever there does not seem to be any great danger of confusing pneumonia. Though the chill and sequent fever are much the same, the cough, pain, rapid pulse, and disproportionately rapid breathing in pneumonia, their absence, together with the presence of pharyngeal redness and the early appearance of the skin-rash, with history of exposure, serve sufficiently to distinguish them. When fine crepitation is present and rusty sputa are expectorated, no room for doubt exists.

Lobar pneumonia is differentiated from malaria by its characteristic temperature, rapid breathing, pain, cough, quick pulse, and the physical signs when present; whilst in malaria the locality in which it usually occurs—more constant, severe, and protracted chill, followed by high fever without marked respiratory trouble, terminating frequently in profuse perspiration, with a tendency to recurrence of chill and fever at fixed periods, and the detection of Laueran's corpuscles—would be conclusive. The spleen is frequently enlarged in malaria.

I have placed but little stress on the presence of the pneumococcus in diagnosis, because I think its value as yet undetermined; and also upon the percussion sounds, because they are so slightly developed in the first stage of this disease.

II.—THE DIAGNOSIS OF CONSOLIDATION OF THE LUNG FROM EFFUSION (SEROUS OR PURULENT).

BY F. FORCHHEIMER, M.D.,
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As between pupil and teacher, no subject is more satisfactory than that of differential diagnosis; as between fellows of a learned society, none less. In the class or lecture-room general outlines are requisite in order that the student may have a diagram which he can fill out; here both generalities and details are filled out, and there remain only such things as can be discussed, or facts concerning which there are varied explanations. It would therefore not be feasible to go into all the points of differential diagnosis that are to be found in my comprehensive subject even if time permitted, since they are most of them described in the text-books so familiar to our students; there will remain, then, only those points which I have found most valuable or most interesting.

In the first portion of our subject, the diagnosis of consolidation and effusion, we will find an easy task if we have to deal only with acute processes running a typical course. It seems almost impossible to mistake a serous pleural effusion for a genuine, typical croupous pneumonia, yet such things are being done daily. The difficulty lies in the atypical acute or chronic cases, and to these I wish to call your attention.

Simon, in a lecture published in the *Progrès Medical* (No. 5, 1891), makes the statement which, to me is rather remarkable, that encysted pleural effusions are quite frequent, and that this encysting may take place either in the upper or lower parts of the chest. If this be so, and my own experience does not lead me to concur in the statement, the differential diagnosis of a pleural effusion will certainly become more difficult. As far as subjective signs are concerned, I believe them to be of little importance, especially in young children; indeed, the sooner the physician accustoms himself to accepting principally those things as facts which he himself observes the better. In both a chronic consolidation and an encysted pleurisy, the

patient has been ill for some time, there has been more or less fever, possibly detected only by the constant use of the thermometer; there has been more or less cough and the general condition of the child may or may not be bad. The dyspnœa would depend largely upon mechanical, local conditions, and might or might not be made of service in differentiation. Who has not seen children running about with appreciable quantities of fluid in their chests, apparently well, with the exception of dyspnœa upon exertion? It is true that they are somewhat emaciated, pale, without much appetite, and, possibly, we may elicit the fact that they sweat profusely during sleep or otherwise. The same may be said for a phthisical consolidation in childhood, and the only result of this examination would be that our attention will be attracted to the respiratory organs as the seat of the trouble. Even this, I take it, will be a great gain, for diagnosis becomes exceedingly difficult in complicated cases of chest-trouble, and, it will be admitted, post-mortem revelations are rather startling in some cases. The books will insist upon one symptom,—the child has had pain. In the majority of instances there can be no doubt upon this score; but where is the pain? How much reliance can be placed on the localization of pain by a three-year-old child? In troubles about the chest it is usually in the epigastric region, and how many affected organs reflect or rather project their sensations to this spot! So that, after all, we must rely principally upon our own examination for diagnosis.

Supposing, now, we are to deal with an encysted pleuritic exudation on the left side, high up, anteriorly. This is evidently a place where differentiation from phthisis becomes difficult; could this differentiation be made? If the quantity of fluid be sufficiently great, there should be very little difficulty; if the quantity be very small, it will be quite, if not altogether, impossible. In the latter instance, few physical signs, if any, will be present to call our attention to the existence of any disturbance (it must be remembered that in a child it takes from fifty to one hundred grammes of fluid to produce evidences of its existence), and if slight indications of trouble be found, the chances are that no one will think of

a small, encysted pleural effusion, and will therefore not make a differential diagnosis, but will come to the more or less warranted conclusion that he is dealing with a small pulmonary consolidation.

If, however, we have evidence of a wide-spread change in the chest, the question might present itself in a different light. A pleuritic exudation will be found, in the great majority of instances, to be secondary to pneumonia, infectious diseases, or to tuberculosis. I do not wish to be understood as saying that there are no primary pleurisies in childhood, but the vast majority of encysted pleurisies are secondary, and secondary to a tubercular infection. If, then, the history does not help us in determining the primary or secondary nature of the affection, we are justified in thinking of an encysted pleurisy.

Inspection of the affected portion may give us valuable information. In the case of a chronic pulmonary infiltration we might or might not have something to record; if any change had taken place, it would have been in the nature of a sinking of that side with all its consequences, rotation of the scapula on that side, possibly slight curvature of the spine, sinking in of the infraclavicular space, and comparative immobility of the part affected. In the case of a larger, encysted collection of fluid, there will, on the contrary, be more or less bulging, and the immobility will not be so appreciable. A sufficient collection of fluid always produces a noticeable change in the contour and size of a child, in our case a localized one, but my experience has taught me that the prominence of the intercostal spaces is by no means the most common form. This will especially be the case in a non-encysted effusion, in which bulging of the intercostal spaces becomes an anatomical impossibility unless the quantity of fluid be very great. Bulging might be possible in the localized form, but we have evidence that is far more valuable, viz., increased resistance upon palpation, felt in the intercostal spaces. This resistance is present even when the child cries, indeed, it is increased by the child's cries. Difficult cases have lead me to lay much stress upon this evidence, and continued observation only verifies its value. Even with retracted intercostal spaces this sign remains, and it is more pronounced in the elastic, thin

chest walls of children than in the adult. This resistance combined with the evidences of percussion are sufficient, in most instances, to differentiate between consolidation and effusion. The percutory evidence is the absence of air, and the distinction between dulness and flatness, when it is possible to obtain this difference. For this purpose it is necessary to percuss very softly, since we frequently percuss for resistance or tension only, as the sounds obtained by percussion in an unruly child are in most cases of very little value. To me, the difference in tension between the side affected and the healthy one is very much greater than the difference between a healthy and a glaucomatous eye. In an encysted effusion, the question of outline of flatness could hardly become a matter of moment, but in a general effusion, the curved outline of the upper level is by no means as characteristic as in the adult, though present at times.

As for change from flatness to resonance, as a result of change in posture in the child, I do not remember having seen it in any case, although it is mentioned by authors as a positive sign.

Auscultation, again, can only be relied upon where the quantity of encysted fluid is great. Even when there is a great quantity of fluid present, the physical conditions are by no means as favorable as in the adult. Sounds are conducted in an apparently most mysterious manner in the infantile thorax; it is only necessary to recall the conduction of bronchial breathing from a pneumonic side to the healthy to illustrate the truth of this statement. If we get no breathing-sounds over the affected portion, we are reasonably sure that a body containing no air is interposed between the ear and the lungs.

This condition can, in the existing case, be expected only over a very limited area, and then can only be detected by using a stethoscope with a small bell. The same can be said for the vocal fremitus, while a diminution can be relied upon in large quantities of fluid; in small quantities the diminution, if any, will be so slight as to escape even the most practised ear.

As for ægophony, I have long ago looked upon it as one of those accidental phenomena which are rarely heard in children,

and therefore of very little practical importance. We are helped along in our differentiation, however, by the positive evidences of consolidation that are usually present,—bronchial breathing, increase in voice-sounds, and possibly the presence of râles. It is by no means positive that a consolidated area will always give us these signs, but an absence will always be temporary, and repeated examinations ought not to fail in clearing up this aspect of the case. However, I have seen so many mistakes made in even apparently so simple a matter as the diagnosis of phthisis, and have made them myself, that I prefer to wait with a diagnosis until repeated examinations have been made. Differentiation would be much easier if, as in the adult, it were possible to examine the sputum in all cases.

Everything being weighed, and the conclusion that we are dealing with a pleural effusion being, tentatively, arrived at, but one thing remains to be done to make the diagnosis positive, if this be found necessary. Small as is the risk of introducing a hypodermic needle into the chest, I do not believe it indicated in order to appease one's thirst for knowledge. A hypodermic needle will, in most instances, determine whether fluid be present or not, but every one will have seen cases in which it is impossible to draw off fluid with a needle when a subsequent operation with a large opening will show that the pleural cavity may be full. It is only recently that a case of this sort has come under my observation: a phthisical colored boy, of eight years, had all the physical signs of a large pleuritic exudation upon the left side, including displacement of the heart and pushing down of the diaphragm and spleen, repeated introductions of the aspirator failed to prove the existence of fluid, yet so sure was I of the diagnosis that thoracotomy was advised. The surgeon delayed the operation for a few days, when intense dyspnœa developing, resection of a rib was done, and it was found that the pleural cavity contained gelatinous, semisolid masses, which could only be removed with the fingers and instruments, there being comparatively little fluid present. In speaking of serous accumulation I have not for the present taken hydrothorax into consideration.

The differential diagnosis of empyema and serous accumulation usually results in a presumptive diagnosis unless the

needle is used. It is true that in some cases presumption almost amounts to certainty, but, from a scientific stand-point, we could hardly place the diagnosis on the same footing as that between a croupous pneumonia and a large, serous pleural effusion. If the term secondary may be applied to any form of pleural effusion, it can be applied to empyema. It is needless to add that by "secondary" I mean that some other disease has preceded the pleurisy. Much may be gained if this fact can be established by the history. However, cases do occur in which an empyema is set up without any preceding previous disease, and, certainly, many serous effusions are secondary. Much has been done to clear up the etiology of pleurisies (Weichselbaum, Fraenkel, Ehrlich, Koplik, and others), but the chapter is by no means finished, and it will be well, for the present, to retain the terms primary and secondary.

I do not believe in the existence of so-called idiopathic inflammations, least of all idiopathic pleurisies; and if even the most careful bacteriological investigators have failed to find a cause in some exudations, it means only that the cause was not a lower form of life but something else.

The age of the patient must be seriously taken into consideration. Seldom do we find serous effusion under four years of age, empyema being most common at that period of life, although exceptions exist to this rule. The thermometer will do something in helping us: where there is pus, we usually have the symptoms produced by accumulations of puss; more or less hectic, diurnal ranges of temperature of a great many degrees, possibly with subnormal temperature in the morning, chills or chilly sensations, sweats, more or less profuse. All these may occur in serous pleurisy, but they are the exception. A long-continued afebrile course certainly speaks against empyema. I have been unable to utilize some of the fine auscultatory signs that have been put down, for instance, the greater transmission of sound in cellular fluids; upon theoretical grounds they may be perfectly correct, but in practice they seem without value, at least in children. To exclude hydrothorax seems an easy matter; it is only necessary to remember that hydrothorax is a dropsy, and the diagnosis becomes clear. There are rare cases in which hydrothorax is

produced by pressure upon large veins or lymphatics, but such cases can be usually recognized by the appearance of œdema in other parts and by chemical analysis of the exudate (Leichtenstern).

After all is said and done, an examination of the fluid is the most reliable evidence we have.

Aspiration either with an hypodermic syringe or with a small aspirator gives absolute evidence in this direction in by far the great majority of cases. The exudation may be so thick as not to pass through the small needle; this fact, as has been stated before, must not be overlooked. Again, cases in which the fluid has separated itself into layers will occur in older children who have been upon their back a long time. In a boy of eleven years who came under my charge in the Cincinnati Hospital, and from whom about fifteen hundred cubic centimetres of fluid were withdrawn, the upper portion of the fluid was perfectly clear, while the lower was cellular. It is necessary to introduce the needle as low as possible in order to get as correct an idea of the fluid as the circumstances permit. The conditions admit no more definite rule, it seems to me, than that the exact location for puncture varies with the patient. For our purpose we wish to get at the lower strata of exudation; when the patient has been forced to lie on his back, the lowest practicable point will be somewhere in the posterior axillary line; when he has been forced to be propped up, it will be between the two axillary lines, and when he has had to sit forward, it will be about the anterior axillary line.

The ninth or tenth interspaces must be chosen for the same reason; the objections that there is danger of going through the diaphragm, its injury, or that the diaphragm may come into apposition with the end of the needle, are, it seems to me, purely theoretical. The only other precaution to be taken is asepsis or cleanliness. It will be conceded on all hands that damage may be done and is being done by the introduction of the needle, but the risk is certainly very small with proper care, though large enough, it seems to me, to make indiscriminate puncture unwarrantable.

III.—DIAGNOSIS OF TUBERCULOUS BRONCHO-PNEUMONIA IN CHILDREN.

BY WILLIAM OSLER, M.D.,
Baltimore.

THE question is beset with difficulties. Broncho-pneumonia is a lesion, not a disease. In the post-mortem room, with the lungs before us, there may be a doubt whether the affection is simple or tuberculous. The following are suggestive points:

(*a*) *The frequency of lung tuberculosis in infants.*—Recent observations have shown that the disease is more common than we have supposed. Leroux's analysis of the statistics of the late Professor Parrot's is particularly instructive. Of two hundred and nineteen cases, in children under three years, there were from one day to three months twenty-three cases; from three to six months, thirty-five cases; from six to twelve months, fifty-three (a total of one hundred and eleven under one year); and from one to two years, one hundred and eight cases. Pulmonary cavities were present in fifty-seven of the cases. Of five hundred autopsies in children reported last year from the Munich Pathological Institute, in one hundred and fifty tuberculosis was present, and in over ninety-two per cent. of these the lungs were involved. The observations of Northrup, at the New York Foundling Hospital, also emphasize the prevalence of pulmonary tuberculosis in early life. These figures show a very much larger percentage than indicated in the older observations of West, Frobelius, and others, and they are of value in calling the attention of practitioners to the widespread existence of infection in infants.

(*b*) *The circumstances under which tuberculous broncho-pneumonia may occur.*—It may develop in a well-nourished infant with healthy antecedents. A case reported by Toulmin from my clinic was very interesting in this respect. The child was aged four months. The family history was irreproachable. The grandparents were living, and the parents were strong and well. When the baby was nine weeks old they moved into a house which had been vacated three weeks before by a

woman who was under our care at the Dispensary with chronic phthisis, and in whose sputa there were numbers of bacilli. In this instance the strong probability is that the child had become infected in the house. The post-mortem showed the most extensive tuberculous broncho-pneumonia, and suppurative and caseous foci in the bronchial glands. The prevalence of infection through the lungs in children is strikingly shown in the recent paper by Northrup. That in every one of one hundred and twenty-five consecutive cases of tuberculosis in children the bronchial glands should be involved tells its own story. The bacilli are inhaled attached to the particles of dust, and reach the bronchial nodes through the lymph-vessels. I have not seen an instance of pulmonary tuberculosis in a child, in which the mediastinal glands were not involved; and how often, too, in autopsies upon children who have died of various diseases, do we find these structures caseous! The constant lodgement of the bacilli at the lymphatic gate-ways, bronchial or mesenteric, is the strongest argument against Baumgarten's theory of hereditary transmission with latency of the germs.

The relation of tuberculous broncho-pneumonia to the infectious diseases has long been recognized. Convalescence from measles, scarlet fever, diphtheria, and whooping-cough is fraught with danger. No one now supposes that the simple broncho-pneumonia, so often associated with these diseases, ever of itself terminates in tuberculosis. When this event happens, it means either a preparation of the soil by the occurrence of catarrhal processes or a lowering of the tissue-resistance, so that the bacilli already existing in the bronchial nodes are enabled to develop. In the latter case the diversion of forces has weakened the defence, advantage of which is taken by an enemy already intrenched but kept at bay.

(c) *Clinical types.*—An acute pneumonic form of tuberculosis in infants is not, I believe, recognized. I have never met with a case in which the onset, the rapid course, and the physical signs simulated exactly acute lobar pneumonia, a simulation of which is by no means uncommon in the adult. The anatomical condition in children is almost always either lobular or pseudo-lobar,—that is to say, the lobe looks uniformly involved; but there are between the areas of consolidation

strands of air containing tissue. Three groups of cases may be recognized. In the *first*, the child is taken ill suddenly during teething or convalescence from an infectious disease. The temperature rises rapidly, and dyspnœa is present with severe cough. The physical signs show areas of consolidation at the apices or central portions of the lungs and numerous râles. Death occurs within a week, or, indeed, within a few days. The post-mortem shows numerous foci of bronchopneumonia; the general appearance of the inflamed areas is red or reddish-brown; only in the cases which persist for a week or ten days are there indications of caseation It is extremely important to bear in mind that some of these cases macroscopically do not present the features of a tuberculous lesion, and, as in an instance reported by Cornil, microscopical examination alone determines the true nature of the disease. The presence of caseous bronchial glands should always excite suspicion. In the *second group* the features are those of a severe inflammation of the lungs. After an infectious disease, or following exposure to cold, the child has a slight cough and gradually becomes feverish. The respirations increase in frequency, the temperature rises to 103° or 104°, the physical signs are well characterized, and no doubt whatever exists as to the existence of a bronchopneumonia. Death may occur after an illness of from three to six weeks. The following abstract from a post-mortem in a case of this kind gives a good illustration of the anatomical condition: "The upper lobe of the right lung is scarcely anywhere crepitant, except at the anterior margin. The middle and lower lobes are heavy and slightly crepitant; the visceral pleura is beset with tubercles which have grown into it from the lung. On section the right upper lobe is occupied with caseous masses from five to twelve millimetres in diameter, separated from each other by an intervening tissue of a deep red color. The middle and lower lobes are stuffed with tubercles, many of which are becoming caseous. Towards the diaphragmatic surface of the lower lobe there is a small cavity the size of a marble. The left lung is more crepitant and uniformly studded with tubercles of all sizes, some as large as peas. There is an acute tuberculous bronchitis in the

smaller and larger branches. The bronchial glands are very large, and one contains a tuberculous abscess." This was from a child aged four months, who had been ill for six weeks.

The *third group* embraces cases in which, following a bronchitic attack, or measles, or whooping-cough, the child has fever of moderate grade and loses flesh. The onset may be insidious, in this way, or, as is sometimes the case, more abrupt and stormy. The physical examination shows areas of defective resonance at the upper and middle portions of the lungs, and moist râles. At first regarded as a case of capillary bronchitis, or broncho-pneumonia, it is only the protracted character of the disease, the supervention of chills and sweats, the gradual development of hectic, the paroxysmal character of the cough, the change in the physical signs indicating softening and breaking down,—these phenomena alone convince the physician that he has been dealing with an instance of pulmonary tuberculosis. I do not here refer to the instances, which are rare indeed, of acute miliary tuberculosis in infants.

(*d*) *The diagnosis.*—In the acute cases we cannot often say whether the affection is simple or tuberculous in its nature. It must not be forgotten that broncho-pneumonia is a lesion, not a disease, a lesion which has a varied etiology, and may be caused by streptococci, by the tubercle bacillus, and possibly by other organisms. The physical signs in the cases of the first and second groups give us few differential indications. The simple and the tuberculous forms occur, perhaps, with equal frequency in the upper lobes. The tuberculous disease is more apt to invade the central portion, and the most marked dulness and physical signs may be at areas corresponding to the roots of the lung. The patchy character of the dulness, the regions of hyper-resonance, sometimes of marked tympany, the blowing or whiffing respiration, with fine râles, are valueless from a stand-point of etiological diagnosis. In the pseudo-lobar form, in which over a considerable portion of one lobe there is dulness or a flat tympany with blowing breathing, it is impossible by the physical examination alone to determine whether the case is one of rapid tuberculous caseation or a lobar pneumonia from cold. In infants under two years lobar

pneumonia is, however, extremely uncommon, while, on the other hand, tuberculous broncho-pneumonia is frequent. Are there any other features in the disease which will help in the differentiation? The fever is not of any value, and, so far as I know, no reliable differences have been determined in the temperature-range of the acute forms of broncho-pneumonia. In the cases following diphtheria and measles, bilateral disease is more common. Enlargement of the spleen, which is present so often, is not of any special value, as it occurs with the fever, and is independent of the nature of the trouble. Perhaps it is more frequent in the tuberculous forms. The physical signs and symptoms failing us, we have to fall back upon the personal history of the child, and of its parents, and the antecedent illnesses. The broncho-pneumonias after measles and whooping-cough are more likely to be tuberculous than those which follow scarlet fever and diphtheria. Naturally, in the latter disease we see a larger number of cases of the streptococcus form than in any other. The case to which I have already referred, a robust infant of four months with healthy parents, shows that even under the most favorable circumstances an intense tuberculous infection may occur.

Practically, then, in cases of the first and second groups, in which the tuberculous broncho-pneumonia is acute and may kill within from three days to six weeks, we are usually unable to say whether the process is tuberculous or not. We can often make a shrewd surmise, remembering that a very considerable proportion of all these cases are tuberculous, but there is no certainty with reference to it.

In the third group, in which the disease is protracted, and in which there are signs of excavation and the child presents really a picture of ulcerative phthisis, the diagnosis is rarely doubtful. Here, too, we have an opportunity, such as rarely happens in the other cases, of examining in the vomit portions of the expectoration and discovering the tubercle bacilli. On several occasions I have been able to do this, and it certainly should be practised whenever possible.

IV.—THE TEMPERATURE IN ACUTE PRIMARY PNEUMONIA OF CHILDREN.

BY L. EMMETT HOLT, M.D.,

New York.

I HAVE endeavored to include among the cases studied statistically only those which were observed throughout their course, since it is only by a study of such records that reliable data are obtained.

Nearly all the cases are from the records of the New York Infant Asylum; a much better place than a hospital for such a study, since the children were under observation at the time they were taken ill. In the fatal cases the diagnosis of pneumonia was confirmed by an autopsy in almost every instance.

Cases of secondary pneumonia occurring with measles, diphtheria, scarlet fever, and gastro-intestinal diseases have been excluded, since in these the primary disease modified very essentially the course of the temperature. A considerable number of cases in which pneumonia developed during pertussis have been included, since this disease played the part of an etiological factor, but did not influence the temperature.

By far the largest number of the cases were under three years, and this fact lends additional interest to this group of cases, since the temperature-curve in the pneumonia of children beyond this age has been well studied by several recent writers, notably by Henry Ashby, in England, C. W. Townsend, in Boston, and Hellström, Jürgensen, and Ziemssen, in Germany. I shall avail myself of some of their figures in order to bring out by contrast the peculiarities in the course of the temperature in children under three years.

I may say, in anticipation of inquiry, that antipyretics have been employed very sparingly in the treatment of these cases, and I do not think that the curves in any of the cases presented have been essentially modified by their use.

Rectal temperatures have, I think, been taken in every instance.

Excluding the classes above specified, there remained for study two hundred and fourteen cases, of which one hundred and sixty-one were cases of broncho-pneumonia and fifty-three cases of lobar pneumonia.

Only one case of broncho-pneumonia was over three years, that one being only one month beyond that age.

Six cases of lobar pneumonia were over three years old. Of the two hundred and seven cases which were three years old or under, one hundred and sixty, or 77.3 per cent., were cases of broncho-pneumonia, and forty-seven, or 22.7 per cent., cases of lobar pneumonia.*

The mortality of the one hundred and sixty-one cases of broncho-pneumonia was one hundred and three, or sixty-four per cent.

The mortality of the fifty-three cases of lobar pneumonia was nine, or seventeen per cent., in every case the pneumonia being complicated. The explanation of this high death-rate is to be found in the fact that these were "institution-children."

LOBAR PNEUMONIA.

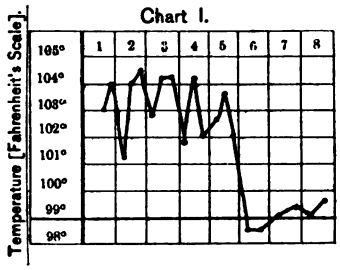
1. *Course of the temperature.*—The typical temperature-curve is exemplified by Chart I., showing a sudden rise, daily fluctuations, usually within the limit of two degrees, with a critical fall upon the fifth day and no subsequent rise.

History.—P. R., male, three years old; in fair condition, sudden onset. Signs of consolidation—bronchial respiration and voice, and dulness—over left lower lobe behind, not distinct until morning of fifth day. On seventh day lung was resolving.

Such typical temperatures were met with in children as young as thirteen months; much more common, however, is it to meet with a remittent type of temperature, and, as a rule, the

* It is interesting to note that the relative frequency of lobar and broncho-pneumonia in children under three years shown in the above figures is almost identical with that found by A. Baginsky (Monograph on Pneumonia, Tübingen, 1880). In Baginsky's cases there were one hundred and fifty-one under three years; of these, thirty-six, or 23.8 per cent., were classed as lobar, and one hundred and fifteen, or 76.2 per cent., as broncho- or catarrhal pneumonia.

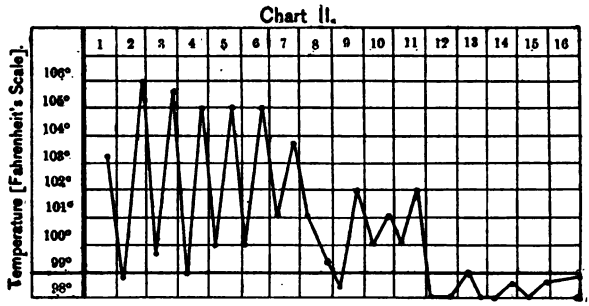
younger the child the more the curve approaches the remittent type. The following case is a remarkable example in which, from the late appearance in the physical signs, the temperature was very misleading. It will be noticed in these cases that,



Typical Curve of Lobar Pneumonia.

although the fluctuations are wide, the temperature very rarely touches the normal, a point which often helps to separate these cases from those of malarial fever.

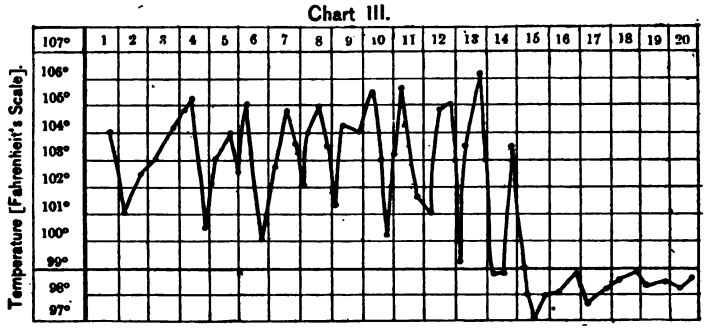
History.—M. M., robust female child, eighteen months old. Sudden onset, no signs in chest till fourth day, then dulness, bronchial respiration and voice at right apex front and back, no râles; crisis on eleventh day, two days after crisis the chest was clear.



Lobar Pneumonia with Remittent Temperature.

In the next case, Chart III., which was evidently one starting as a central pneumonia, the remittent temperature is also shown. The fluctuations in this case were wider in the latter part of the illness, the reverse of what was seen in Chart II.

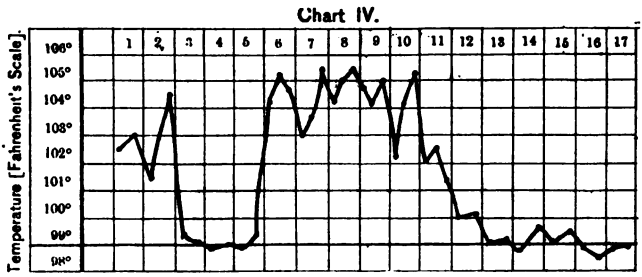
History.—A. D., female, in fair condition; sudden onset; repeated examinations of chest made, but no abnormal signs until ninth day. Very rude respiration and slight dulness at right apex, front; on twelfth day all the signs of consolida-



Lobar Pneumonia with Remittent Temperature.

tion at the same point, no râles; four days after crisis, lungs were clear.

The following case illustrates one of the more uncommon ways in which a lobar pneumonia may develop. There is seen a sudden onset, with symptoms of an usual attack of pneumonia, which subsided after two days; then after three days of complete absence of fever the disease again started up in the



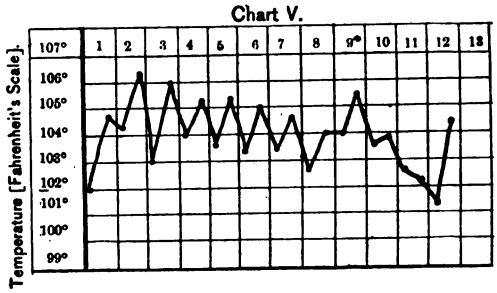
Abortive Pneumonia in Left Lung followed by Typical Lobar Pneumonia in Right Lung.

opposite lung. This ran its usual course, the temperature falling by lysis on the fifth and sixth days, and thereafter was normal.

History.—W. I., male, seventeen months, healthy; sudden onset; on second day disseminated fine râles in both lungs behind, and over left lower very feeble respiration, high pitched, *i.e.*, some bronchitis with congestion (?) of left base. On third, fourth, and fifth days, general symptoms gone and signs nearly disappeared. On sixth day all symptoms of pneumonia and on seventh distinct consolidation of right base, rest of chest clear. Subsequent course typical, resolution rapid and complete.

In the fatal cases of lobar pneumonia the steadily high temperature was the predominating type.

Chart V.



Lobar Pneumonia Fatal.

History.—A. R., female, two years old, robust; sudden onset; signs of consolidation on the third day in right upper lobe. Autopsy showed gangrene of lung; very extensive gelatinous pleural exudate containing in its meshes five to six ounces of pus. Consolidation of right upper and middle lobes.

TABLE I.

Course of Temperature in Lobar Pneumonia. (Recovery Cases.)

Steadily high temperature	12
High, with remittent type	22
Medium temperature, with moderate fluctuations	9
Total	43

2. *Mode of termination of the fever.*—The fever in lobar pneumonia more frequently terminates by crisis than by lysis, as shown by the following table:

TABLE II.

Showing mode of Termination of the Fever in Three Hundred and Forty-two Cases of Lobar Pneumonia under twelve years which ended in recovery.

	Lysis.	Crisis.
Pendlebury Hospital	25	201
Townsend, C. W.	8	29
Hellström	5	25
Holt	25	24
Total	63	279

The difference in the proportion of my own cases ending by lysis (fifty per cent.) and the general proportion (eighteen per cent.) is very striking. It is to be explained, I think, by the difference in age. The observations of the writers quoted have been made upon older children, and fully ninety per cent. of their cases of pneumonia occurred in children over three years old, while of my own cases all but six were under this age.

Possibly I may have been a little more stringent in the use of the term "crisis," as I have included only those in which the fall to normal was steady and occupied less than twenty-four hours.

The critical day of the cases above referred to was mentioned in three hundred and thirty-nine cases; it was as follows:

TABLE III.

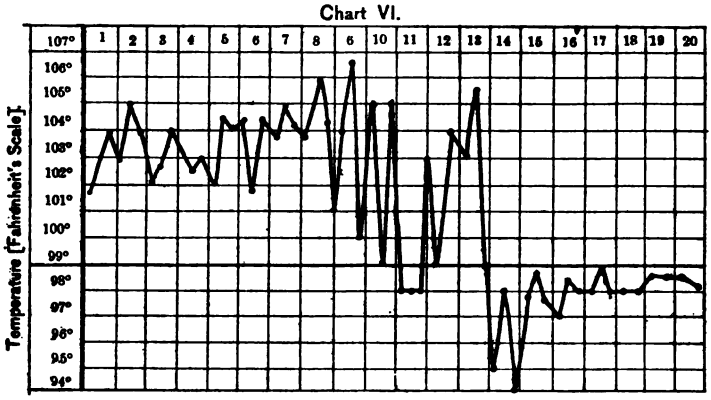
Showing Critical Day of Three Hundred and Thirty-nine Cases of Lobar Pneumonia in children under twelve.

Crisis on 2d day	2 cases.	Crisis on 10th day	17 cases.
" " 3d "	8 "	" " 11th "	7 "
" " 4th "	28 "	" " 12th "	6 "
" " 5th "	47 "	" " 13th "	4 "
" " 6th "	60 "	" " 14th "	6 "
" " 7th "	73 "	" " 18th "	3 "
" " 8th "	50 "	" " 21st "	1 "
" " 9th "	32 "		339 "

It will be seen from this table that the most frequent critical day is the seventh; and that in two hundred and thirty of the cases, or about sixty-eight per cent., the fever ended between the fifth and the eighth days.

At the time of the crisis the temperature not infrequently falls below the normal. The following case (see Chart VI.) illustrates this point to an unusual degree; here the tempera-

Chart VI.



Lobar Pneumonia; Recovery.

ture fell in the course of fifteen hours 10.5° F., from 105.5° to 95°; again, later in the same day after a slight reaction, it fell to 94°, with marked general symptoms of prostration, and it was not until two days had passed that the normal temperature was maintained. This is seen to a less degree in a very large number of the cases.

History.—L. C., female, nineteen months old, fairly healthy; sudden onset, symptoms typical but physical signs delayed; consolidation in left mammary region on eighth day; on ninth in right lung middle lobe; on tenth day pseudo-critical drop, followed after twenty-four hours of apyrexia by a further rise, which was accompanied by signs of extension of disease in right lung. Resolution rapid after crisis.

The most frequent causes of post-critical rise are two; first, an extension of disease to a new area in the lung, and, secondly, the development of complications. Those by far most frequent among my cases have been pleurisy with the production of a great amount of fibrin, and empyema.

It will be seen that in several of the cases reported that the crisis, even when it was marked, has been delayed considerably beyond the average period, as shown by Table III. A study

of the cases shows very clearly that this is very much more likely to be the case with children under three than among those who are older.

Thus the duration of the fever in forty-two recovery cases (all but six of which were under three years) was over seven days in twenty-five, or sixty per cent. While among three hundred and thirty-nine cases, over three years, the duration of the fever was over seven days in but one hundred and twenty-six, or twenty-seven per cent.

3. *The highest temperature and the mortality.*—The highest recorded temperature in the fifty-three lobar cases was as follows:

TABLE IV.

Highest Temperature.	Recovery Cases.	Fatal Cases.
107.5° F.	0	2
106.8°	1	0
106.5°	1	0
106.°	4	8
105.5°	8	1
105.°	12	2
104.5°	6	0
104.°	7	0
103.5°	8	1
103.°	2	0
	44	9

It will be seen that of the nineteen cases in which the temperature did not pass 104.5°, but one died,—a mortality of five per cent.

Of thirty-eight cases in which the highest point touched was 105.5°, four died,—a mortality of ten per cent.

Of eleven cases in which the temperature rose to 106° or over, five died,—a mortality of forty-five per cent.

It would appear, then, that a temperature which reaches only to 104.5°, or thereabouts, may be considered normal to the disease, and certainly not one which calls for treatment, *per se*. Further, that above 105° the mortality rises with the increase of each degree of temperature.

Summary.—1. The temperature-curve in lobar pneumonia of children over three years old resembles the adult type, being steadily high and terminating by a crisis, this occurring in nearly eighty per cent. of the cases.

2. The most frequent critical day is the seventh; although it occurs almost as often on the sixth, eighth, and fifth days, the frequency being in the order named.

3. Under three years the proportion of typical cases is very much smaller; crisis occurs in only about one-half the cases, and this is usually at a later day than among older children.

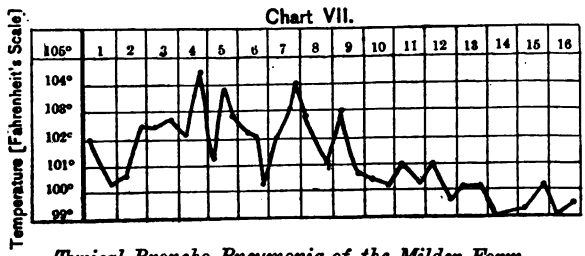
4. Under three years the temperature tends towards the remittent type, wide fluctuations being not uncommon in uncomplicated cases. In a smaller number it is steadily high until the crisis.

5. The mortality is in direct proportion to the height of the temperature when it is above 105°, below this point it seems to bear no constant relation to it.

6. The most frequent causes of a post-critical rise or of delayed crisis is extension of the disease in the lungs or complicating pleurisy.

BRONCHO-PNEUMONIA.

1. *The course of the temperature.*—There cannot be said to be any typical course followed by the temperature in broncho-pneumonia in the sense in which that term is used with reference to lobar pneumonia. The great majority of the cases, however, fall quite readily into a few groups, examples of which are here given. There are not a few, however, which are so irregular that they resist all attempts at classification.



Typical Broncho-Pneumonia of the Milder Form.

In Chart VII. we have a good example of a rather mild case with a gradual onset, and a termination in recovery.

The constitutional symptoms were not grave and followed the course of the temperature.

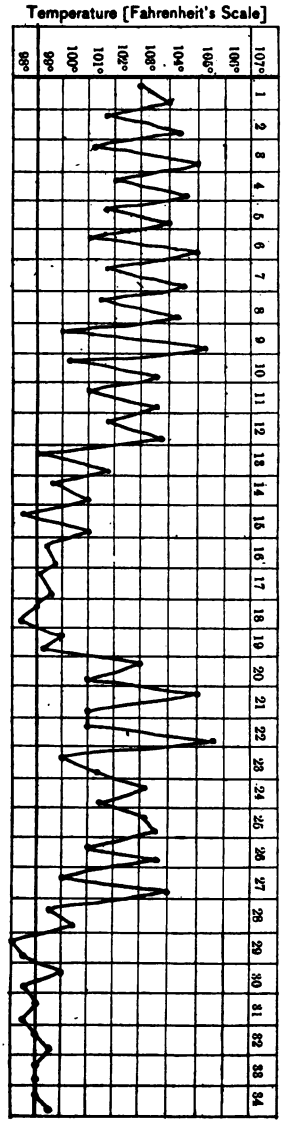


Chart IX.

Relapsing Broncho-Pneumonia; Recovery.

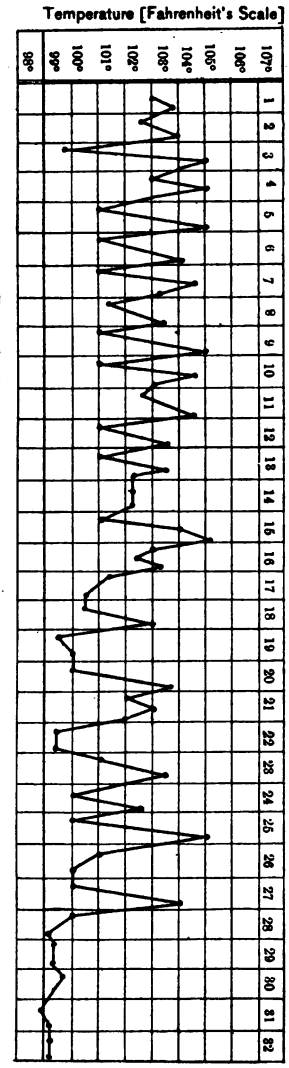


Chart VIII.

Broncho-Pneumonia; Prolonged Course; Recovery.

History.—H. D., male, sixteen months old; delicate child; previous bronchitis; onset gradual, signs of consolidation at

left base on fifth day, but fine râles heard in both lower lobes behind. Resolution was slow, râles persisting for a long time in both lungs behind.

The next case (Chart VIII.) illustrates a longer course of the disease, with an irregular, remittent temperature, rarely touching the normal line. This variety was seen in quite a large number of cases, and the duration varied usually between fourteen and twenty-eight days.

History.—M. B., female, eighteen months, in fair condition; sudden onset. Early signs were localized, fine râles over left base; on fifth day signs of consolidation here with râles on both sides behind. General symptoms of moderate severity. Signs of consolidation disappeared about a week after cessation of fever, râles persisted nearly two weeks longer.

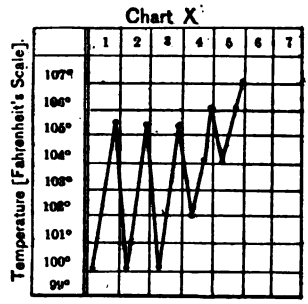
The next temperature chart (IX.) is that of a relapsing pneumonia or a recurrence before complete resolution. The first attack was a fairly typical one, the acute symptoms lasting about two weeks, and then, while resolution seemed to be progressing satisfactorily, there was a new accession of fever, accompanied by new signs in the chest, and a second attack followed, shorter and milder than the first. Sometimes the interval between the two attacks is two or three weeks, sometimes longer still. There is always a possibility of such an occurrence until the lung has quite recovered, as shown by the disappearance of all physical signs. In some cases two or three such recurrences have been seen in a single season.

History.—J. B., male, nineteen months; delicate. Consolidation on sixth day in left lower lobe behind, two days later small area of consolidation in right lower lobe behind; many râles both sides; eighteenth day, signs of consolidation had disappeared, but many râles persisted. Accession of fever on nineteenth and twentieth, accompanied by extension in disease as shown by new râles, but no evidences of consolidation during second attack. Slow resolution and convalescence.

Chart X. is a type of a large number of fatal cases in which the temperature, after two or three oscillations, mounts steadily until death takes place. Not infrequently, the duration of these cases is only two or three days, and death often takes place before any signs of consolidation in the lung are found.

The inflammatory process is so intense and the prostration so great that they include a large number of the very worst cases met with.

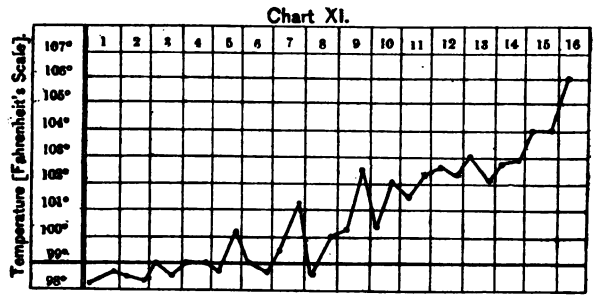
History.—J. B., male, six months old, markedly rachitic; sudden onset. Signs first day were fine moist râles throughout the chest, marked prostration and cyanosis; on third day, a



Broncho-Pneumonia; Fatal.

small area of consolidation at the upper third of left behind; increasing prostration, cynosis, and death.

Autopsy.—No pleurisy, consolidation at left apex behind, and posterior two-thirds of left lower lobe; consolidation of right apex posteriorly, lower lobe intensely congested.



Fatal Broncho-Pneumonia following Bronchitis.

The next case (Chart XI.) shows the gradual development of pneumonia following bronchitis in a patient with pertussis, the temperature gradually rising as the disease extended from the larger to the smaller bronchi, and finally to the alveoli of

the lung. The development in many cases is more rapid, but the same type is preserved.

History.—J. M., six months, male; delicate; pertussis for three weeks. Early signs only of bronchitis of large tubes; on eleventh day signs of consolidation in right upper lobe. Increasing prostration, death cyanosed.

Autopsy.—Large areas of consolidation in right middle and upper lobe, small scattered spots throughout left lung.

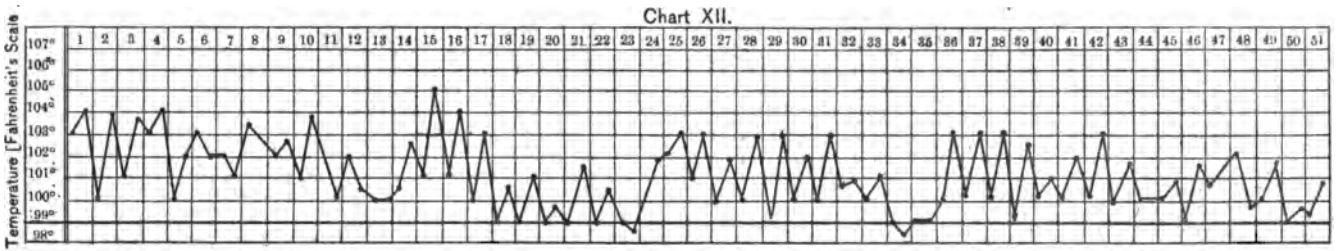
The following case is of unusual interest on account of prolonged course of the disease, the duration being over seven weeks of continuous febrile symptoms. Without an autopsy, the diagnosis of tuberculosis might have been urged with some reason. This error in diagnosis is, no doubt, very often made in similar cases. This case progressed, as most such cases do, by a series of exacerbations; a distinct one is shown by the temperature to have occurred on the fifteenth day; another on the twenty-fifth, and still another on the thirty-sixth. The general symptoms were at no time very intense, the temperature most of the time being below 103°, and death taking place from exhaustion due to the gradual extension of the disease (Chart XII.).

History.—S. H., male, two and a half years, healthy; sudden onset; for the first two weeks the only signs were very fine moist râles throughout both lungs front and back. The râles in front in great part gradually cleared up; those behind persisted, but it was not until the thirty-fourth day that positive signs of consolidation were discovered in the left lower lobe behind; these signs gradually extended, and before death were present over nearly the whole left lung behind and over the right lower lobe. There were also friction sounds over both sides.

Autopsy.—Old and recent pleurisy with general adhesions; left lower lobe completely solid; patches of consolidation in left upper lobe. Right lower lobe about one-half consolidated, with patches elsewhere. Bronchial glands large, but not cheesy. No evidence of tuberculosis either upon gross or microscopical examination.

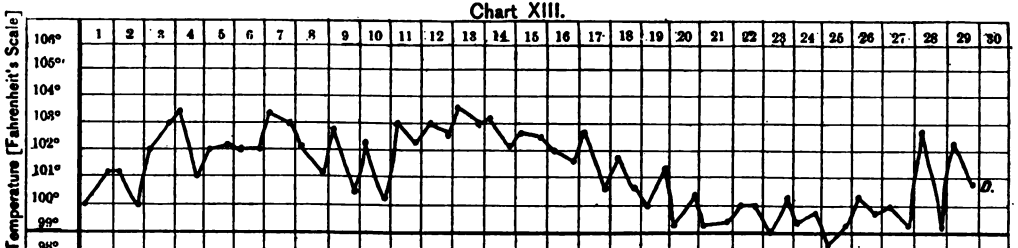
In nearly every one of the charts thus far there is seen in the temperature a more or less distinct morning remission and even-

Chart XII.



Fatal Persistent Broncho-Pneumonia.

Chart XIII.



Fatal Broncho-Pneumonia, with regular Evening Remissions and Morning Exacerbations.

ing exacerbation. Occasionally, without any cause that can be discovered, we find the temperature higher in the morning than it is in the evening, for several days together. The following case (Chart XIII.) is in this respect a most extraordinary one, since throughout the whole course of the disease, with the exception of a very few days, the temperature was regularly higher in the morning than in the evening. The case in other respects was fairly typical, with its three weeks of fever, never very high; then nearly a week in which the temperature was scarcely above the normal, but with no other signs of improvement; finally, an acute exacerbation and death in two days.

History.—W. P., male, seven months, in fair condition; pertussis for three weeks; gradual onset; on the seventh day consolidation at the left base behind. In a few days the greater part of the left lower lobe was solidified and a small area was found in the right base. General symptoms severe though temperature was low. Repeated attacks of cyanosis and collapse, and death from exhaustion.

Autopsy.—Typical broncho-pneumonia, with consolidation of nearly the whole left lower lobe and scattered areas throughout the right lung. No pleurisy.

In all the charts thus far a temperature of 104° has been reached in every instance, and in most of the cases this point was touched repeatedly. Broncho-pneumonia, even the acute primary form, may run its course with a temperature very much below this point. Such cases are particularly obscure ones for this reason. Persistent low temperature is seen oftener among infants under eight months, and more frequently in the very delicate than among those of robust constitution. In such cases the temperature may never be above 101°. I have met with one case in which the highest temperature was 99.5°, and the greater part of the time it was subnormal; yet the illness lasted over two weeks and the physical signs were typical. The child was a Cretin and of very feeble vitality.

The following table gives a general idea of the relative frequency with which the variations in the temperature were met with:

TABLE V.

Course of Temperature in Broncho-Pneumonia.

	Fatal Cases.	Recovery Cases.
Steadily high	19	...
High and remittent	40	21
Low, early, and high late	8	...
Moderately high (ranging from 101° to 104°)	15	14
Low (generally below 103°)	17	15
Very low (generally below 101°)	4	...
Subnormal	1	...
	104	50

2. *Mode of termination of the fever.*—In broncho-pneumonia this is almost invariably by lysis; a definite crisis is quite a rare event.

3. *Duration of the fever.*—The exact duration of the febrile period in one hundred and fifty-six cases is shown in the following table:

TABLE VI.

Duration of Fever in One Hundred and Fifty-six Cases of Broncho-Pneumonia.

	Recovery Cases.	Fatal Cases.	Total.
2 days	...	9	9
3 "	...	8	8
4 "	3	6	9
5 "	...	9	9
6 "	2	3	5
7 "	6	8	14
8–10 days	6	19	25
11–14 "	13	9	22
15–21 "	12	20	32
22–28 "	3	8	11
29–35 "	3	5	8
35–42 "	...	6	6
51–60 "	2	1	3
	50	106	156

This table may be condensed so as to bring out more forcibly the relation of the duration of the fever to the mortality, as follows:

TABLE VII.

	Cases.	Deaths.	Mortality.
Whole number	156	106	68 per cent.
Duration of less than 4 days.	12	12	100 "
" 4 to 7 days.....	37	26	70 "
" 8 to 14 "	47	28	60 "
" 15 to 21 "	32	20	62.5 "
" 22 to 35 "	19	13	68.4 "
" over 35 "	9	7	77.7 "

It will be seen that the highest mortality is among the cases of shortest duration, all of those proving fatal that were shorter than four days. In these cases the child is usually overpowered by the violence of the disease, which produces death usually by acute congestion of the parts of the lung not previously involved.

If this early danger is passed the chances of recovery are much improved, since, during the period covered by the next four days, the mortality falls to seventy per cent.

The lowest death-rate of sixty per cent. is seen in the cases terminating in from eight to fourteen days. If the disease continues beyond two weeks the chances of recovery are lessened every day the fever lasts, so that beyond five weeks more than three-fourths die.

4. *The highest temperature and the mortality.*—The following table shows the highest recorded temperature in one hundred and sixty-one cases of broncho-pneumonia.

TABLE VIII.

Highest Temperature in One Hundred and Sixty-One Cases of Broncho-Pneumonia.

Temperature.	Recovery Cases.	Fatal Cases.	Total.
108.° F.....	...	4	4
107.5°	...	2	2
107.°	...	7	7
106.5°	...	7	7
106.°	5	15	20
105.5°	9	5	14
105.°	15	28	43
104.5°	10	7	17
104.°	9	12	21
103.5°	2	2	4
103.°	5	2	7

	Recovery Cases.	Fatal Cases.	Total.
102.5°	1	3	4
102.°	1	4	5
101.5°	0	1	1
101.°	1	1	2
100.°	...	2	2
99.5°	...	1	1
	58	103	161

The general mortality of the cases was therefore sixty-four per cent.

Of twenty cases in which the temperature reached 106.5° or over, the mortality was one hundred per cent.

Of seventy-seven cases in which the highest point was 105°, 105.5°, or 106°, the mortality was 62.4 per cent.

Of forty-nine cases in which the highest point was between 103° and 104.5°, the mortality was forty-seven per cent.

Of fifteen cases in which the temperature did not reach 103°, the mortality was eighty per cent.

We learn to be apprehensive not only in cases of very high temperature in broncho-pneumonia, but also in cases with abnormally low temperature. This latter class of cases is a very fatal one, not, however, because the temperature is low, but because of the circumstances under which such low temperatures are to be met with, viz., very early infancy, feeble vitality and great prostration with little reactionary power.

Summary.—1. The predominating type of temperature in acute primary broncho-pneumonia is high and remittent, the daily fluctuations amounting usually to from three to five degrees, F. The sustained high temperature is uncommon except in the rapidly fatal cases. A low range of temperature only two or three degrees above the normal is not very uncommon, but is more frequent in fatal cases.

2. The termination of the fever is almost invariably by lysis.

3. The lowest mortality is among cases in which the fever lasts from eight to fourteen days; the highest is among those lasting but two or three days, and next to these the protracted cases lasting over four weeks.

4. The day of highest temperature in fatal cases is usually the last day; in recovery cases there is no rule in this respect.

5. The lowest mortality is seen in the cases in which the highest point reached by the temperature was between 103° and 104.5° F. Above 105° the mortality rises with the increase of each degree in the temperature. Abnormally low temperatures are also to be dreaded, since they usually indicate a constitutional condition which makes recovery very doubtful.

V.—THE DIAGNOSIS OF ACUTE BRONCHO-PNEUMONIA FROM BRONCHITIS.

BY L. EMMETT HOLT, M.D.,
New York.

THERE is no doubt that these two diseases have been long confounded, not only by practitioners but by medical writers. The more careful microscopical study made in the last ten years of the lesions in the acute pulmonary diseases of young children has placed it beyond dispute that a very large number of cases classed formerly as "acute bronchitis" are really cases of broncho-pneumonia. This applies to nearly the whole group of cases formerly described under the heading of "capillary bronchitis." In infants, it seems almost impossible for an acute inflammation of the fine bronchi to occur without the process extending to the alveoli which surround them, unless death takes place at a very early stage in the disease.

The diagnosis of bronchitis of the large tubes from broncho-pneumonia ordinarily presents very few difficulties: the coarse character of the râles, the low temperature, and the absence of the symptoms of serious illness usually make this unmistakable. Again, there are many cases in which it is just as evident that pneumonia exists, both from the severe general symptoms and the physical signs which indicate a solidification of some part of the lung.

It is the class of cases lying between these two which we shall make the special topic of discussion. In regard to them, it may be said that though in the largest number the diagnosis can be made with tolerable certainty, in many it is difficult, and in some it is impossible. It may be urged that, as there

is no difference in the management of a case of severe bronchitis and one of broncho-pneumonia, a diagnosis is of no practical importance. This may be admitted from the standpoint of the therapeutist in the acute attack. It is, however, essential, to a correct understanding of what so often follows such an attack,—relapses, recurrences, or chronic pneumonia,—to know that from the beginning there existed not merely bronchitis but broncho-pneumonia.

The main points of differentiation of acute bronchitis from acute broncho-pneumonia may be grouped under three heads: the difference in temperature, in the severity of the general symptoms, and in the character of the physical signs.

First, as to temperature: Acute bronchitis commonly begins with a rise to 102° or 103°; usually after twenty-four hours have passed it falls to 100°, and remains between this point and 101.5° for several days, gradually reaching the normal.

If a case begins with this initial temperature supposed, and the fever rises on three or four successive days to 102.5° or 103°, it is almost certain that something more than bronchitis exists. If the fever is due to disease of the lungs, we may be reasonably sure of pneumonia. On the other hand, the existence of a temperature below that supposed does not preclude the possibility of pneumonia. In my series of cases, I have met with several of acute primary pneumonia, even fatal cases, with a temperature scarcely above 101°. Such must be diagnosticated by the other symptoms,—prostration, cyanosis, etc.,—and the physical signs.

A continuous high temperature for a number of days, then, of pulmonary origin, warrants us in diagnosticating pneumonia rather than bronchitis, but a low temperature does not exclude pneumonia. The general symptoms—cough, prostration, restlessness, dyspnœa, and cyanosis—are all, as a rule, less severe in bronchitis than in broncho-pneumonia. But it is the general severity of all, rather than anything particular about each one, that is significant. The more severe they all are the greater the probability of pneumonia.

The physical signs of consolidation, dulness, bronchial breathing, etc., are conclusive evidences of pneumonia, when

the symptoms themselves are doubtful. Absence of such signs, however, does not exclude pneumonia, even though they are never obtained throughout the attack. This cannot be too strongly emphasized. Scores of times I have found quite large patches of broncho-pneumonia at autopsy, when on the day previous the most careful examination had failed to disclose any bronchial respiration, bronchial voice, or dulness. The explanation was that the pneumonic areas were so separated by healthy pulmonary tissue that at no point was there enough hepatization to produce any of the ordinary signs by which consolidation is recognized. These are really the only difficult cases, but, unfortunately, they are very frequently met with. Inspection, palpation, and percussion are of little or no value whatever.

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The auscultatory signs to be considered are pleuritic friction sounds, the character and localization of the râles, and the nature of the respiratory murmur. Pleuritic sounds may be looked for in broncho-pneumonia whenever we have large areas of consolidation present, but in my experience based upon post-mortem observations, and it has not been small, almost never under other circumstances. In the cases under discussion they are so rarely heard that they may then be dropped from our thought altogether.

Again the character of the râles does not help us much, for the reason that in broncho-pneumonia it is the *bronchitis* and not the *pneumonia* which produces most of the abnormal sounds.

The localization of the râles is of more value. Unilateral bronchitis that is only bronchitis is of very doubtful existence. I do not think I have ever seen it. I refer now to cases in which an acute inflammation remains confined to the tubes of one side; cases are not uncommon in which the signs are unilateral for the first few hours. The signs of localized bronchitis of the finer tubes give us sufficient reason for diagnosticating pneumonia in doubtful cases, provided generally that tuberculosis may be excluded. Localized subcrepitant râles over both lower lobes behind are especially to be looked on as denoting pneumonia. Simple bronchitis is commonly a bilateral disease, both sides being symmetrically

affected, and the anterior as well as the posterior parts of the chest.

There are cases of disseminated broncho-pneumonia which give rise to generalized subcrepitant râles over the whole chest. These are the cases described by many of the older writers under the term "capillary bronchitis;" all of these are, I believe, cases of broncho-pneumonia. The severity of the general symptoms—dyspnœa, cyanosis, and prostration—are sufficient usually to distinguish them from cases of simple bronchitis.

In conclusion, we make the differential diagnosis between bronchitis and broncho-pneumonia by weighing both the symptoms and the physical signs, and of the two the symptoms, as a rule, are the more valuable. But if we regard every doubtful case as one of pneumonia, we will make very few mistakes, for such the doubtful cases almost invariably turn out at the autopsy to be.

15 East Fifty-Fourth Street, New York.

DISCUSSION.

Dr. Rotch.—We would like to hear from Dr. Jacobi.

Dr. Jacobi.—I came in but recently, and will wait till the others have spoken, and then may make a few remarks after I shall have heard the discussion.

Dr. Koplik.—It was my good fortune during the past two years to make the diagnosis in many cases and to see them at the operating table, both of serous and purulent effusions in children ranging from six months in age to as many years. And what interested me much was the question of tuberculosis and encysted effusions. Whereas it is very common in tuberculosis to find masses tying down the lung and causing thickening, it is not so very rare to find encysted structures in pleurisies of pneumonic nature. I have seen at the operating table a skilful operator open the chest and find very little where he had supposed a large effusion existed. When the finger was introduced, it was found that it entered a cavity formed by adhesions; upon breaking up this cavity a second one was entered, where a large amount of effusion existed, and this effusion examined, turned out to be purely pneumonic, and a good recovery was made; so that the encystment of an exudate is common in a perfectly acute effusion.

As to the results of percussion, there is something about the resistance to the percussion finger that is characteristic when a chest is distended with fluid. If anything is atypical, the physical signs of pleuritic effusion in children are, and we can put reliance on none of them. There is in all cases local dorsal flatness and a loss of fremitus when the child cries. In these cases, if a needle is introduced, the diagnosis of effusion is confirmed. We cannot put any reliance in the broncho-vesicular breathing or other classical signs. As to the postponed crises, when crisis does not occur between the eighth and thirteenth day, I suspect other processes. No matter how small the affected area may be, it may be a pleurisy with effusion. Under these circumstances, it is justifiable to put a needle into the chest if there is any difficulty in the breathing.

DR. ROTCH.—I would like to ask Dr. Jacobi if there are not many cases in which the disease lasts forty or fifty days?

DR. JACOBI.—As I arrived late, I preferred to listen to what the other gentlemen had to say, and then to take part in the discussion, but as the President has twice asked me to speak, I will say that as to the indefiniteness of the time when, in many cases, resolution sets in there is no doubt in my mind. In speaking of the earlier stages of croupous and catarrhal pneumonia we are too schematic. We speak as if we always had a well-defined class of pneumonias. There is many a case that does not run its course in this schematic way. It has been my fortune to see cases that were not complicated at all, but quite often do we find croupous pneumonia with pleurisy, lobular pneumonia with pleurisy, croupous pneumonia with interstitial pneumonia, etc. Hybrid forms are more often met with than we usually admit; thus it is best explained why the course of pneumonia is frequently not so regular as we expect. The question has been asked whether it is my opinion that a shorter or longer period than twelve days occurs before crisis. We have all seen cases that lasted only a few days, crisis occurring in two or three days, but croupous pneumonia has a well-developed course in from seven to ten or fourteen days, and in such the crises will be natural I will answer affirmatively that many cases will exhibit the so-called regular time. We have heard of so-called intermittent pneumonia. Now, I do not believe malaria has anything to do with it. There are cases of engorgement that run their course in two or two and one-half days and terminate favorable, and may then break out again in a few days, when again we behold a number of days with exudation and positive symptoms of pneumonia. These are the cases that we call intermittent or

malarial. When we do have actual malaria, it is an accidental combination. After all, then, I am convinced that we may have pneumonias which run their course in more or less than schedule time. It has been remarked there are many complications, and why not? In genuine croupous pneumonia we have an exudate in the alveoli which coagulates readily. In catarrhal pneumonia we have a change in the larger bronchi, alterations of the epithelia, secretion of mucus, etc. There we have to deal not so much with coagulative as with a catarrhal and epithelial change. In a great many cases we find in pneumonia the complication with pleurisy or with interstitial changes. There can hardly be a case of pneumonia in which the interstitial tissue does not participate; if the latter predominate there is dulness and fever from the beginning, as in an uncomplicated case; but it may last weeks or months, and finally we have lysis and convalescence. This may be perfect, but very often the absorption is not completed, the interstitial process terminates in the formation of new tissue. These are the cases which result in retraction of the pulmonary tissue and dilatation of the bronchial tubes. Those pneumonias which last long are often complicated with interstitial inflammation, and that is why croupous pneumonia lasts so long either actually or apparently, and finally gets well. As far as crisis and lysis are concerned, I do not think that they are always well pronounced. Lysis is not so marked as it might be. It is often slow. There is this difference between croupous pneumonia of adults and of children; there is in the latter lysis as often as there is a crisis. That makes the diagnosis of genuine pneumonia more difficult than in the adult. There are other causes why the diagnosis is difficult. No doubt the gentleman has gone over the ground fully and in every particular. The simple cases ought not to be considered here. What should occupy this Society is the exceptional cases in which the diagnosis is difficult.

DR. ROTCH.—The symptoms of the early stage of lobar pneumonia would be gladly received from Dr. Jacobi.

DR. JACOBI.—If you would allow me to make a few more random remarks, the Society is aware that it is the President's suggestion and not mine. If I were asked whether I had a positive symptom in diagnosticating every lobular or interstitial pneumonia, I should say "No." In a plain, simple case every one will probably diagnosticate pneumonia on the second day, but probably not often on the first day with certainty. There is dyspnœa, accentuation of expiration after the second, third, or fourth day, fever and occasional vomiting, which, however, we may find in a number of other diseases. It is

only after a time that the diagnosis can be made. There are other reasons why the diagnosis is difficult, expectoration is seldom or never seen. It may be bloody, or it may not, and in a well-established case, too, but we do not see the bloody sputum. The child swallows it. Besides, there are cases in which it is impossible to locate the pneumonia. It has taken me sometimes three and four days to locate a pneumonia beginning in the centre. Frequently, when there are complications, there are a number of other symptoms which are confusing. The cerebral symptoms, which give us the impression that we have a meningitis, cannot be properly diagnosticated until after a few days have elapsed. In a number of cases in which the diagnosis of meningitis was made, I made a diagnosis sometimes by an accurate examination of the lungs, but now and then just as positively by examining the prominent symptoms. In meningitis we are not likely to find the quickened respiration of pneumonia. The simple fact that the number of respirations was unduly increased made me certain that I had to do with meningitic symptoms developed by pneumonia. I have not seen a case of pneumonia in which the rate of respirations was not increased unless the meningitic symptoms predominated over the respiratory. I have tried more to speak of the difficulty of making a diagnosis than to indicate how to make the positive and accurate diagnosis in any easy case. As we have no sputum, examination of this can be of no use. If we had it, even microscopical examination would not be positive. For Friedlander's coccus is not always found in croupous pneumonia; on the other hand, it may be met with in lobular pneumonia and other diseases, and is found outside the lungs. Fränkel's coccus is found in lobular pneumonia also, so this examination would not benefit us, and I am not in a position to give a microscopic sign concerning any of the forms of pneumonia. Perhaps it is good to speak of one point that may be known to everybody. There is one test that will indicate pneumonia in many a doubtful case; that is the careful examination of the axilla. Always strip the child and percuss gently in both axillæ. Many times a slight dulness will be found here when nowhere else. But percussion must be very gentle; strong and forcible percussion is always out of place. It may be permissible on the chest of an adult, but only gentle percussion will do on the chest of a child. Dr. Northrup spoke of bronchial respiration; in a large number of cases we do not have it, but we have bronchophony. That is what the crying of a baby is good for. A pneumonic child that says nothing deprives us of this symptom, but when he opens his mouth to scream, the

difference of the voice on the right and left sides is diagnostic, and has helped me in many cases where the diagnosis could not be made otherwise.

DR. SMITH.—I think the gentlemen must have been surprised at the number of croupous pneumonias under the age of three years seen by Dr. Holt. I think, when he prepares his paper, he should state the grounds upon which he has made the differential diagnosis between croupous and catarrhal pneumonia, the latter being the common form of pneumonia in patients under the age of three years. A high temperature beginning abruptly, continuing three or four days and then abating, does not certainly denote that the pneumonia is croupous. It may be that in such cases there is a condition of the digestive organs that aids in causing this elevation of temperature. It used to be said that in the fatal cases a fibrinous exudation in the alveoli indicated croupous pneumonia, but it is now known that there is sometimes a fibrinous exudate in the alveoli in catarrhal pneumonia.

DR. HOLT.—The symptoms on which the diagnosis was based were the physical signs of circumscribed consolidation, sudden onset, continuous fever, and rapid resolution. There was one question I wished to raise. Has it happened to others, in cases of pneumonia complicated by purulent meningitis, to be surprised at the absence of cerebral symptoms? In two of my cases these were so slight as to be almost overlooked, yet the autopsy showed a most intense meningitis.

DR. FORCHHEIMER.—Meningitis is common in adults, and was present in five out of the hundred cases in which I made autopsies. As a rule, where cortical effusions occur, there are most marked symptoms. No one can tell when he has only cortical symptoms, which are no more than those of congestion without any exudation. Of the pleurisies, more than one-half have marked meningitis of the convexity.

DR. SMITH.—I referred to the relative frequency of croupous and lobar pneumonia as well as to the symptoms.

DR. FORCHHEIMER.—If Dr. Holt would give us the post-mortem appearances in these cases, we would be better pleased. It is surprising that one person should see so many cases in infants under three years of age. In my more limited experience I have seen few cases of lobar pneumonia in children. There is a pseudo-lobar pneumonia affecting many lobules, with only narrowly separated alveoli, but that is lobular and not lobar.

DR. JACOBI.—There was a time when children were believed to have a pneumonia of their own, and adults one of

their own. Adults had a patent right of lobar and children had the same of lobular. I remember to have read lately a statement that almost every case of pneumonia in a child was lobar as in the adult, but I think I can say that only children of eight, nine, or ten years begin to be adults as far as their pneumonias are concerned. Children of three and four have lobular pneumonia. Babies of a few weeks are apt to have lobar pneumonia, and until they are five or six weeks old I have seen more lobar than catarrhal, but after that the vast majority exhibit the lobular form. About one-third of those I have seen at those ages were lobar and two-thirds lobular. I should have liked to listen to more remarks from Dr. Forchheimer, and I think this Society would also like to hear from the President.

Dr. Rotch.—Some years ago I reported a case of lobar pneumonia dying on the second day of life, and the diagnosis was verified by the autopsy. I agree with what Dr. Jacobi has said regarding the relative frequency of pneumonia at different ages, and I believe that the time is coming when we shall be able to explain the reason for its so occurring on histological grounds.

Dr. Holt.—In making up my tables, I first put quite a number of cases in a doubtful class; but after examining them critically again, I put the doubtful cases in the catarrhal class, leaving only those in the lobar class,—only those that were clearly characterized by sudden onset, sharply defined physical signs of consolidation in one lobe or part of a lobe, and rapid resolution. These were, in brief, the symptoms upon which I based the diagnosis, and if they are not conclusive, I would like to know how we are to make the diagnosis of lobar pneumonia. The proportion of lobar cases under three years was twenty-five per cent.

Dr. Jacobi.—Those who see many babies soon after birth say that in the first two weeks of life we see more lobar than lobular pneumonia. There is Dr. Northrup, who sees so many at the Foundling Hospital, and Dr. Smith. I would like to hear the experience of those who see many babies in the first few weeks of life.

Dr. Northrup.—There is a tradition at the Foundling Asylum that all lobar pneumonias get well. I must indorse Dr. Holt in all he has said except regarding the proportion, as to that I do not know. After three years the pneumonia has a right to be of the adult variety; between two and three a small proportion may be lobar, but it is seldom fatal. I can nearly number on my fingers the fatal cases I have seen.

DR. SMITH.—Many of the catarrhal pneumonias get well also, and their duration varies from two to sixty days, at least that has been my experience.

DR. FORCHHEIMER.—I wish to call attention to the fact that this question was worked out in 1835 by Dr. Gerhardt, whose papers on pneumonia remain to-day as good as any written on the subject. His views quite coincide with those of Dr. Smith.

DR. ADAMS.—I have been reading on the subject, and think there is much in climatic surroundings. In going over the records for seventeen years in the Children's Hospital, lobar pneumonia is the exception. Dr. Acker and I had a discussion as to this point, and, in an experience since 1875, I said I had not seen any croupous pneumonia, and for that reason I thought it singular. I went over the records, and there were only three or four cases recorded of croupous pneumonia, and I do not think there can be any mistake in regard to this point, as the records were the result of examinations by Drs. Busey and W. W. Johnson. I am rather surprised at the number of croupous pneumonias reported. Catarrhal pneumonia is common, and the mortality is not so high, so I think, if this large percentage of lobar pneumonia is found in New York, it must be due to climatic conditions. During the epidemic last year, though I hesitate to refer to this oft-discussed grippe, there were a number of cases in regard to which I could not make up my mind whether it was lobar pneumonia or certain undefined symptoms that occurred, and there were many such in children under five years of age, to which I prefer to give some other name.

DR. JACOBI, on invitation of the President, on the second day, said: Before commencing, I wish to remark that Dr. Huber was prevented from being present by the sudden illness of a sister. He would have certainly been here but for this misfortune.

I was very sorry for having been absent when the papers were read last evening, and that is why my remarks were not in so good a shape as they should have been before such a society as this; thus I forgot to add one single item that may be of importance in many cases. I tried to confine myself to cases in which the diagnosis is difficult,—in which even an expert would be tempted to make an incorrect diagnosis. I allude to the complication that may arise from the presence of anomalous glands. We know to what extent the mediastinal glands may be affected, and to what extent they are either the cause or the result of sub-acute bronchitis. In a number of cases in which auscultation and percussion is our only guide, the

presence of large quantities of mediastinal glands leads to confusion. Not infrequently will they give rise to dulness, either anteriorly or posteriorly, or both. Anteriorly, I have met them mostly on the left side; posteriorly, between the scapulæ near the hilus. Like tubercular infiltration of the lungs, a glandular mass will result in diminished respiratory murmurs by compressing the lungs. Sometimes, when arranged near large bronchi, they will produce brochophony, or bronchial respiration. This effect may be obtained as well near the diseased lung as near the healthy one. Thus an actual difficulty of diagnosticating—at least the extent of—a pneumonia may result from the presence of large glandular masses.

HOW TO PREVENT COMPLICATIONS AND SEQUELÆ IN SCARLET FEVER.

BY J. LEWIS SMITH, M.D.,

New York.

THE type of scarlet fever varies greatly in different epidemics and in different years. In the time of Sydenham it was a mild disease, being seldom fatal, but a few years later its type changed, and it became one of the most fatal maladies of early life. The late Dr. Graves, of Dublin, said that at one time the Dublin physicians congratulated themselves on the fact that they were so successful in treating scarlet fever, losing very few patients, while statistics showed that it had been very fatal in the practice of their predecessors in Ireland. They believed that their better success in treatment was due to a better understanding of the malady, and a better use of therapeutic agents. By and by the type changed, the death-rate largely increased, and the Dublin physicians learned to their sorrow that the good results of the treatment of scarlet fever, which they had attributed to their knowledge and skill, had been due to the mildness of its type. Several years ago a prominent New York physician treated over fifty cases of scarlet fever in an institution without a death, and he made the remark that he thought that he had achieved the mastery

over this disease. In a few months the type of the malady changed, and his own child, a bright and promising boy of four years, was one of the victims.

Although the type of scarlet fever is at times very mild and its death-rate low, and considerable improvement has been made in the last few years in the employment of measures designed to prevent as well as to cure it, nevertheless the deaths resulting from it continue large. In New York City the fatal cases from this malady in 1887 were five hundred and eighty-nine, in 1888 were thirteen hundred and sixty-one, in 1889 twelve hundred and forty-two, and in 1890 four hundred and eight. In Brooklyn during the same years the deaths from scarlet fever aggregated twelve hundred and forty-six.

Whenever an epidemic of scarlet fever presents a severe type, deaths occur in not a few instances directly from the intensity or malignancy of the disease. The system is overpowered by the profound blood-poisoning. Malignant scarlet fever begins, as is usual, without premonition, but with a very high temperature, and a rapidity of pulse corresponding with the temperature. Restlessness and delirium ending perhaps in convulsions and coma supervene.

In these cases suddenly overpowered by the intensity of the scarlatinous poison, the eruption has a dusky red color, the circulation especially in the extremities is slow or languid, and the extremities may be cool, while the internal temperature is at or above 105°. In epidemics of a severe type many patients with these symptoms die within the first week, even within the first three or four days, with the best possible treatment.

But there is no other disease of childhood in which so many fatal complications occur as in scarlet fever, changing at once a favorable to an unfavorable prognosis; and I am induced to prepare this paper in the belief that the physician can by judicious treatment and a correct understanding of the indications prevent some of these complications, or render them milder, and thus be instrumental in saving life.

Intense inflammation of the fauces, followed by gangrene of the faucial surface, cervical adenitis, cellulitis, and perhaps an

abscess, inflammation of the Eustachian tube, even with gangrene along its surface, and severe, protracted, painful, and dangerous otitis media are frequent in epidemics of scarlet fever of a severe type, we will endeavor to show that complications which greatly increase the gravity, suffering, and death-rate of scarlatina may be rendered much less frequent by timely and proper preventive treatment, treatment consisting of the early and frequent application to the nasal, post-nasal, and faucial surfaces of non-irritating germicide remedies.

Fränkel and Trendenberg, and Professor Hutinel, of Paris, may be mentioned among those who recommend the early and frequent application of disinfectants to the fauces, as a means of diminishing the severity of scarlet fever, and preventing complications. Professor Hutinel says, "The micro-organism of scarlet fever is not yet isolated, but now we know the germs that are found in the lesions produced by the usual complications of the disease. In nearly all the cases of adenitis, arthritis, nephritis, endocarditis, and pleuritis from scarlet fever, the bacteriological examination has shown a streptococcus that is very much like, if it is not the same, the Rosenbach pyogenic streptococcus." This is, adds Hutinel, constantly seen in the pharynx when the disease begins, and it constantly plays an important part in the genesis of the inflammations seen there (*Archives of Pediatrics*, September, 1890). Professor Hutinel has charge of the pavilion for the treatment of the scarlatinal patients connected with l'Hôpital des Enfants Maladies, and of course has an ample opportunity for observations. In the *Revue Mensuelle des Maladies de l'Enfance*, July, 1886, Fränkel and Trendenberg relate the results of their microscopic examination of three cases of scarlet fever that died when the eruption was passing off. They found large numbers of streptococci in different organs, and these organisms presented the peculiarities of the streptococci of pus. The number of micro-organisms corresponded with the intensity of the inflammation. These observers found abundant micrococci in the lymphatic glands in the immediate vicinity of the pharynx. They state that their observations teach them that, in order to prevent secondary infection, the physi-

cian must vigorously treat the local changes in the pharynx and adjacent parts from the beginning of scarlet fever.

Recent bacteriological investigations in reference to scarlet fever, therefore, have shown that microbes occur abundantly upon the inflamed faucial, nasal, and post-nasal surfaces in this disease, and that the one which closely resembles it, if it be not identical with that which occurs in suppurative processes, is the most abundant. Moreover, in the adenitis, cellulitis, and other internal inflammations which supervene as complications or sequelæ of scarlet fever, organisms occur which are apparently pathogenic, and seem to be identical with those occurring upon the faucial surface. The microbe commonly found is the streptococcus. Therefore, the theory that pathogenic organisms occur abundantly upon the inflamed faucial and nasal surfaces in scarlet fever, and that more or fewer of them enter the system through the capillaries or lymph-channels, and cause the internal inflammations, which complicate this disease, receives support from recent investigations. Even if the advocates of this theory have exaggerated the facts, we cannot, I think, fail to see, in the present status of our knowledge, that early and frequent disinfection of fauces and nares in scarlet fever, whether mild or severe, should be insisted on not only for the purpose of diminishing the inflammation of these parts, but of preventing, so far as possible, the more serious internal complications. The best mode of applying this treatment appears to be spraying or irrigation with an antiseptic, as the peroxide of hydrogen, one part to four of water for the fauces, one part to eight of water for the nares, used hourly or every half hour, or of corrosive sublimate, two grains to the pint of water, employed every two hours, within non-poisonous limits or with some other non-irritating but efficient disinfectant. A nasal injection should always be warm. I have used as an antiseptic wash for the nostrils, with apparent good result, a combination of boracic acid, sodium borate, and common salt, as in the following formula:

℞ Acidi boracici, ʒii;
Sodii borat., ʒii;
Sodii chloridi, ʒi;
Aquæ puræ, Oi. Misce.

One teaspoonful to be injected into each nostril hourly.

Cold applications along the sides of the neck, as muslin frequently wrung out of alcohol and ice-water in cases attended by a high temperature, afford some aid in reducing the pharyngitis, and preventing cellulitis and adenitis, but they are much less efficient for this purpose than the antiseptic sprays, which destroy the microbic cause of the complications.

Eclampsia is always a very dangerous complication. It occurs either early in the disease, when the type of the scarlet fever is severe and the temperature dangerously high, or in the declining stage of the disease when its cause is usually uræmia. Eclampsia occurring late in scarlet fever, or after the latter has ceased, and being, therefore, a sequel, is a symptom of nephritis, and we will postpone its consideration until we treat of the renal disease, but eclampsia in the first days of scarlet fever is due to the severity or malignancy of the malady, which early and profoundly affects the nervous system, and is very fatal, so that its prevention is a matter of importance.

Eclampsia thus occurring is certainly at the present time with the new remedies provided by modern chemistry, and abundantly tested by clinical observations, a much more preventible complication than in former times. In the first years of my practice with the use of the old remedies, eclampsia, occurring during the first three or four days of malignant scarlet fever, was much more frequent and fatal than it has been of late years with the appliances of modern therapeutics. Recognizing the fact that the convulsions occur in those whose temperature is dangerously high, we should regard the reduction of the temperature by safe and efficient methods as an important indication for the prevention of eclampsia, as well as of other dangerous nervous manifestations, as delirium or coma.

Most recent writers on scarlet fever recommend as an efficient means of reducing heat the use of cold water externally in one form or another, either by itself or combined with a spirituous lotion or vinegar. Trousseau, commenting on the good results obtained by Currie, of Liverpool, in the beginning of the present century, from cold effusions in scarlet fever, proceeds to speak of the strong popular prejudice against the use of cold in the disease, and continues, "Very well, then, in place of giving your patients cold effusions give them

mere lotions of water at 25° C. (77° F.) . . . Let the patient be placed on a folding-bed, and then let the entire body, first the anterior and then the posterior surface, be rapidly wetted with sponges soaked in this water, . . . following the same rules, as after cold effusion,—that is, returning the patient to bed and wrapping him in blankets without being dried." Trousseau speaks of the benefit resulting from such treatment in diminishing the "extreme aridity and stinging heat," the skin becoming cooler and moist, and the cerebral symptoms diminishing. Von Ziemssen recommends the immersion of the patient in water, at the temperature of 90°, and the gradual addition of cool water until the temperature of the bath falls to 77°. In a few minutes the patient is returned to bed and covered with bedclothes, when his temperature will be found reduced two or two and a half degrees. If the patient complain of chilliness, or his pulse be feeble, he should be immediately removed from the bath and whiskey or brandy administered, for, if the extremities remain cool and the capillary circulation sluggish, collapse may occur, or some internal inflammation may arise. Ziemssen, like other physicians of experience, has discarded the use of cold water in adynamic cases, when the pulse is weak, the surface dusky, and the capillary circulation sluggish, but uses instead hot or warm water with alcohol to the extremities.

Professor Henoch, in his treatise on diseases of children, published by Wood, says, "These apparently threatening cerebral symptoms are undoubtedly due to the continual high fever. . . . If the fever continue high and the apparently malignant symptoms, described above, develop, the head should be covered by an ice-bag, . . . and the child placed in a lukewarm bath not under 25° R. (88.25° F.) I decidedly oppose cooler baths in scarlatina, which presents a tendency to heart-failure; cold may produce an unexpected rapid collapse, more than in any other affection." In his more recent treatise, published by the New Sydenham Society, Henoch reiterates his objections to the use of baths in scarlet fever at a lower temperature than 88° F., and states that on several occasions he has known collapse to occur while patients were in the bath, and in one instance death. Similar remarks on the use of cold

baths or bathing for the purpose of reducing the heat in hyperpyretic scarlatina, and relieving the restlessness, jactitation, and delirium, premonitory of eclampsia or coma, might be quoted, did time permit, from other standard writers.

Aided by the experience of such men as Trousseau and Henoch, are we able to formulate a method of employing water as an antipyretic in scarlet fever, and thereby preventing dangerous nervous complications or symptoms, which we can confidently recommend to the profession. In all hyperpyretic cases of scarlatina, whether its form be sthenic or asthenic, accompanied by pronounced nervous symptoms, an ice-bag or its equivalent, a linen or silk handkerchief, wrung out of ice-water every five or ten minutes, should be constantly applied over the head, so long as the temperature remains at or above 103°. The ice-bag should be about one-third full, so that it fits over the head like a cap. If a handkerchief be used, the popular objection to the use of cold may be in a measure overcome, by adding one-fifth part of alcohol to the water, or, as Henoch recommends, adding vinegar to it. At the same time as a potent means of abstracting heat, at least when the temperature is at or over 104°, a similar application should be made around the neck, and especially along its sides. Cold applications over the great vessels of the neck, the jugulars and carotids promptly abstract heat from the blood, while it diminishes the pharyngitis, adenitis, and cellulitis, as we have stated above. In sthenic cases, in which the extremities have a pungent heat, a bright red color, and active circulation, the limbs should be frequently sponged with cool water containing alcohol or vinegar. If the temperature with this treatment be not sufficiently reduced, the hands and forearms may be immersed in the lotion, while the patient is still in bed, or a double thickness of muslin or linen, frequently wrung out of ice-water, may be placed upon the hands and arms. This treatment is grateful to the patient, is not attended by any shock, and continued two or three hours, it usually reduces the temperature two or more degrees.

In asthenic cases, with a dusky color of the skin, a sluggish capillary circulation, coolness of the extremities, or a pungent heat, cool applications, although beneficial when applied to the

head and neck, are likely to be injurious if applied to the extremities. On the other hand, in these asthenic cases the frequent application to the extremities of tepid or hot water, with brisk friction, as recommended by Ziemssen and others, accelerates the flow of blood, revives the functional activity in the torpid limbs, and is evidently useful. Allowing the hyperpyretic patient to hold in the mouth and swallow pieces of ice has been a common practice for a long time, and is very agreeable. I would also recommend for hyperpyretic patients as a means of reducing heat, especially when emesis is present, and the quantity of nutriment retained is insufficient, a clyster every third hour of ice-cold peptonized milk, containing one of the sarco-peptones. With proper precautions and discrimination of cases, the use of cold in the ways which I have mentioned for the purpose of reducing hyperpyrexia, is, I think, safe, provided that no renal complication exists.

Quite a large number of antipyretic drugs has been used in scarlet fever, but most of them have been adversely reported on by distinguished clinical observers, either because they are inadequate or are dangerous remedies, and in some cases harmful, and have therefore fallen into disuse, or are not commonly prescribed. Henoch says, in regard to the treatment of really malignant cases, "I have never seen any successful result from the use of large doses of quinine, internally or subcutaneously, and I regard salicylate of sodium, as well as antipyrin and antifebrin, as remedies which are dangerous in such cases, and may favor the occurrence of collapse." I may add to Professor Henoch's condemnation of salicylate of sodium that it sometimes produces albuminuria, and may be a factor in causing nephritis. Professor von Ziemssen, while he recommends hydro-therapeutic treatment of scarlet fever, expresses the opinion that antipyretic drugs are of secondary importance. Reimer and others might be mentioned, whose remarks in regard to these agents are similar to those of Henoch and Ziemssen. Reimer, in the large institution with which he is connected, employed experimentally less known remedies, as kairin and thallin, but reports adversely on their use. Veratrum viride, the American hellebore, was for a short time employed in this country, at least in New York,

in hyperpyretic scarlet fever; but those who, like myself, prescribed it, subsequently discarded its use, since it is dangerously depressing and is liable to cause collapse. Notwithstanding these adverse opinions, there is, I think, sufficient evidence that two antipyretic drugs are useful in certain cases, so that they can be confidently recommended to the profession for the reduction of the temperature when dangerous nervous symptoms, as restlessness, jactitation, and delirium are present, which are forerunners of eclampsia or coma. These medicines are aconite and phenacetin. Neither of them should probably be given in cases of extreme malignancy, characterized by feeble pulse, dusky color of the skin, and sluggish capillary circulation; but in hyperpyretic cases not markedly adynamic or malignant, they are safe and useful if properly employed.

In his instructive paper on the treatment of scarlatina, read before the American Pediatric Society in 1889, Dr. Fruitnight stated that he had records of sixty-three cases of scarlet fever treated with aconite, with only three deaths. He had employed antipyrin and antifebrin in a considerable number of cases, but abandoned them on account of the symptoms of cardiac depression, and sometimes of collapse, which they caused. Dr. Fruitnight gives a table of cases, showing the reduction of temperature produced by the tincture of aconite root. The average adult dose of this medicine is about three minims every three hours. A child of eight years could take one-third, and one of twelve years one-half, of this dose. I have also observed a good result from phenacetin administered in half-grain doses to a child of eighteen months, and in one-grain doses to a child of the age of three to five years, every two or three hours, with an alcoholic stimulant. We repeat that in cases attended by marked depression, phenacetin should not be prescribed, or it should be prescribed in small doses, its effects be carefully watched, and an alcoholic stimulant be employed at the same time.

Although, as Professor Henoch has stated, a high temperature in itself produces nervous symptoms, restlessness, jactitation, delirium, somnolence, and that fatal complication eclampsia, and we recognize the importance of employing antipyretics,—modern therapeutics has discovered a drug, not

regarded as antipyretic, which exerts a wonderful sedative effect on the nervous system, relieving dangerous symptoms, and which can be safely administered in large and frequent doses. We refer to the bromide, either of potassium or sodium. Prescribed in doses of five grains every hour or second hour for a child of three to five years, it will often, according to my observations, control threatening nervous symptoms and produce quiet rest.

In malignant cases, with frequent, rapid, and weak pulse, ante-mortem heart-clots are liable to occur, constituting a fatal complication. To prevent this, cardiac tonics and stimulants should be employed, as digitalis, musk, camphor, the carbonates, or aromatic spirits of ammonia, and the alcoholic stimulants. These agents produce stronger contraction of the cardiac muscular fibres, and thus diminish the danger of the formation of thrombi. Ammonia, administered in large and frequent doses to young children, is very liable to cause gastritis. To prevent this, it is well to administer it in milk, or after milk has been taken. Three grains of carbonate of ammonia should be given in half a wineglassful of milk every hour or half hour to a child of five years suffering from heart-failure. I am aware that both Henoch and Vidal speak disparagingly of carbonate of ammonia in these cases, regarding it as too weak, but my observations, and the favorable observations of others, induce me to continue its use, when there are evidences of heart-failure.

Musk, in doses of three-fourths of a grain to three grains every two hours, was at one time considerably prescribed as a cardiac stimulant, but it is apparently inferior in its action to camphor. Camphor should be prescribed in doses of one to two grains every two hours. It may be employed hypodermically, as recommended by Henoch, dissolved in five times its quantity of rectified spirit, and the same quantity of water. Alcohol, whether administered in one of the stronger wines, as sherry, or in whiskey or brandy, is also a most useful and, indeed, indispensable cardiac stimulant.

The so-called scarlatinous rheumatism, whether or not a true rheumatism, is probably of microbic origin, being produced by the scarlatinous germ or by a secondary organism. In itself

it involves little danger, but the endocarditis or pericarditis, which sometimes occurs in connection with it, is a serious complication. So far as we understand its nature, it does not seem improbable that it may be prevented or rendered milder, and especially the pericardial or endocardial inflammation, by the germicide treatment, which we have recommended for the prevention of other complications.

Finally, cannot that common and fatal complication and sequel, glomerulo-nephritis be rendered less common or less severe by appropriate preventive measures? We have stated elsewhere that the fact that this inflammation occurs, in so many instances, in the declining stage of scarlet fever, when there is reason to think that microbic agency is slight, and in the mildest cases that are carelessly treated, incline clinical observers to the belief that the common cause of the nephritis is taking cold, by too early and injudicious exposure to draughts of air, or by out-door life. Hence the cautious and experienced physician directs his patient to remain in a warm room, with the avoidance of currents of air, during the period of desquamation, or until two or three weeks have elapsed after the disappearance of the rash. This is advice in which I fully concur.

But distinguished bacteriologists and pathologists, whose opinions on most subjects are accepted by the profession, believe that the renal disease, though sometimes produced by taking cold, is more commonly caused either by the direct action of "the specific virus of scarlatina, or by some complicating secondary organism" (Professor Welsh),—in other words, that its cause is microbic. This theory receives support from the investigations of Babes, if the statement made in medical journals be correct, that he has found secondary microbes in kidneys in thirteen or fourteen cases of scarlatinal glomerulo-nephritis. Hence, in the present state of our knowledge, it is evident that the physician, in endeavoring to employ such measures as will minimize the liability to the renal affection, must recognize the two theories relating to its causation. He will give orders that his patient, during two or three weeks of convalescence, or the period of desquamation, shall remain in a room constantly at a temperature of 70° to 75°, and avoid

FROM A PHOTOGRAPH OF A CASE OF TUBERCULAR OSTEITIS OF THE HIP.

draughts of air. He will also employ from his first visit the local antiseptic treatment recommended above, for the purpose of destroying, so far as possible, "the specific virus of scarlatina" and "secondary organisms," which may in certain cases be the chief factor in producing the glomerulo-nephritis.

DISCUSSION.

DR. FRUITNIGHT.—I wish to allude to one point. I have noticed that scarlatinal nephritis occurs most frequently in mild cases of scarlet fever. On account of this mildness, good care is not taken of such cases. They are apt to be neglected or even ignored altogether. Therefore, as a rule in my practice, in every case, be it mild or severe, I always impress upon mothers and nurses three of these cardinal principles of treatment: 1, Warmth; 2, Rest; 3, A milk diet. By following out these principles, sequelæ are usually prevented.

A CASE OF TUBERCULAR OSTEITIS OF THE HIP ORIGINATING IN COLORADO.*

BY JOHN M. KEATING, M.D.,
Colorado Springs; Vice-President American Pediatric Society, etc.

THE following is the history of a case I have recently seen in consultation with Dr. James A. Hart, of Colorado Springs; it is so interesting that I have taken the liberty of placing it on record. The patient is a remarkably well-developed boy of six years, born at Colorado Springs, an altitude of six thousand feet, with a very bad family record. Surrounded by all the comforts and luxuries that wealth can give, living an open-air life which the wonderful climate affords, he develops a tubercular joint-affection, probably through direct inheritance, and forms a seeming exception to facts most interesting which investigation has brought out.

Dr. Hart's notes read thus:

"B. G., born in November, 1885, at Colorado Springs, where the family had come from Ohio, in 1882, on account of the

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* Read by Dr. Watson.

health of the father. The father died of consumption in 1887, and probably was in an advanced stage of the disease when the child was conceived. The father's parents both died of phthisis, his maternal grandfather died of cancer of the stomach.

"The child as an infant was fairly healthy; when about three years of age he was taken to Europe, and while there developed a general eczema which resisted treatment, and which did not disappear until he reached home. He also while away was troubled with indigestion and diarrhœa.

"In September, 1889, while in Europe, the mother first noticed the child limping and complaining of pain in the ankle; this in a short time disappeared, in a measure, but in the following year he complained of pain above the knee, and it was noticed that he 'favored' the limb in walking, and that he 'stooped in pulling on his stocking.'"

Examining the case in April, 1891, Dr. Hart located the trouble in the right hip-joint. The boy was sent to New York to Dr. V. P. Gibney for treatment. As will be seen by the photograph, the boy is at present in an excellent condition of health, his nutrition seems perfect, and the apparatus he wears enables him to play out-doors daily.

It may be added that the boy has two sisters, one seven and the other nine; each one is strumous,—one having enormously enlarged tonsils with enlargement of the lymphatics in the neck.

In reply to a note I wrote to Dr. Gibney, he sent me the following:

"My views of the B. case are simply these. The lesion is an osteitis in the epiphysis and possibly in the diaphysis. There is no assurance that a focus does not exist in the acetabulum. The joint should be protected at all hazards, and the result then ought to be good,—that is, he ought to get well without any shortening and with function restored. In reply to your questions.

"1. There is little danger of stiffness of the knee (from the splint) resulting, such stiffness is contrary to rule. When it does occur, a little use soon breaks it up and a good knee is the result.

"2. I care nothing about stiffness (knee) or muscular atrophy. I am bent on protecting this *hip* against trauma, whether from muscular spasm or from injuries external to the body.

It is sometimes desirable to increase muscular atrophy in order that the reflex spasm can be better controlled.

"Believing as I do that the process is nearly always tubercular; convinced, as we all are, of the extreme difficulty of managing tubercular processes; knowing the clinical history and its results of tubercular lesions about the hip, even under what is called good treatment, I feel like omitting nothing that will contribute to the well-being of this particular hip."

As will be seen, Dr. Gibney's report removes all question as to the case being other than one of tubercular disease; indeed, the history of the case, its invasion, its symptoms, are in themselves sufficient evidence.

I find, upon inquiry, that a large number of the twelve thousand people of Colorado Springs are tubercular; that intermarriage among the so-called cases of *cured* tuberculosis (and I regret to say many not so pronounced) is by no means infrequent, and yet I can obtain the histories of but five cases of tubercular osteitis of the hip occurring within fifteen years. Indeed, I believe that there are on record, after most careful search, but eight or ten cases of *acquired phthisis* in persons resident there without previous taint. If the bacillus of Koch be as rapacious as it should be, considering the immense field it occupies, certainly many more cases would be added to our list, and, therefore, I cannot but believe that our little patient is not a victim of a bacillus rampant, but has received his infection direct from his father, which lay dormant in his lymphatic tissues until possibly a slight traumatism produced a focus of irritation in the epiphysis of his right femur, and there determined the process. I have, as an insurance man, always been very much interested in the questions of heredity, and though I am ready to give full credit to the bacillus tuberculosis as a causative factor in the transmission of disease, yet I cannot but feel that there is a something, besides receptivity, which is transmitted from parent to child that when properly nurtured will produce the disease. Some physicians of large experience have thought that tubercular meningitis in children is more frequent in Colorado than elsewhere, that is to say relatively so, but this is a very difficult matter to decide, when we consider the immense concentration of the tubercular population

in the State. On the contrary, it certainly seems from the statements made that, whether due to altitude, dryness, sunshine, porous soil, or all combined, *there is a decided immunity from tuberculosis among the child population at Colorado Springs, which is even apparent in those subjected to the most baneful influences of a tubercular ancestry.*

But the matter of cure in such cases as the one herein narrated has always presented a very chaotic appearance to my mind. Why should absolute rest, such as is outlined in Dr. Gibney's letter, render further inroads of the bacillus futile, not, indeed, finally secure its death and the patient's recovery? Until I read the admirable paper by Dr. W. Watson Cheyne, in the *Lancet* for November 15, 1890, I did not fully understand the subject of the expectant treatment.

His views are that "tuberculosis in man is essentially a local disease. In the struggle between the tissues of the human body and the tubercular virus, the opposing forces are pretty well matched, and the victory does not always rest with the bacillus." That many cases recover without any operative interference whatever,—in other words, that rest—surgical rest —will limit the bacillus to the focus of primary irritation, and it will die without further dissemination. Indeed, in a conversation upon the subject, recently, Dr. Newton Shaffer, of New York, expressed to me the same views, concluding that in all probability the determination of the bacilli to the joint, as to the lung, was possibly brought about by traumatism or irritation, however slight, and that by the expectant treatment adopted at an early stage, especially if the focus be in the epiphysis, as is usually the case, the joint being rendered immovable, the bacillus would be confined to the point of invasion by the bony structures of the shaft, and would be deprived of its power of doing further harm.

Although I am not aware of the percentage of cases of tubercular hip-disease in general, certainly five cases in fifteen years, in a population averaging five thousand individuals annually, of whom possibly *one* in *seven* is tuberculous (C. F. Gardiner), strikes me as remarkably low, and probably warrants the drawing the lesson from this study that immunity from inherited and acquired tuberculosis of other parts as well as

the lungs can come from proper care and hygienic surroundings.

A patient under such treatment should then be subjected to climatic influences as are known to be productive of the best results in restraining the evil effects of the bacillus, and these are purity of atmosphere and plenty of sunshine.

A CLINICAL STUDY OF ONE HUNDRED AND FORTY-TWO CASES OF HEART-DISEASE IN CHILDREN.*

BY FLOYD M. CRANDALL, M.D.,
New York.

THIS study is based upon one hundred and thirty-five cases of acquired cardiac disease in children under fourteen years of age, and seven cases of congenital malformation. For about one-third of these I am indebted to Dr. Holt, who has kindly placed at my disposal the record books of the Northwestern Dispensary.

Of these, fifty-one, or thirty-eight per cent., were boys; eighty-four, or sixty per cent., were girls. Of the congenital cases four were boys and three were girls. Their ages are shown by the following table:

Years.....	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
Males.....		1	2	1	4	6	4	1	8	6	5	6	6	1	— 51
Females..		1	3	4	7	9	10	8	9	12	14	3	1	3	— 84
Total...		2	5	5	11	15	14	9	17	18	19	9	7	4	—135

Hence less than nine per cent. occurred under five years, while thirty-eight per cent. occurred under eight years. The ratio of males to females for the whole time was one hundred to one hundred and sixty-four; during the first eight years it was one hundred to one hundred and eighty-eight, and for the next seven years one hundred to one hundred and fifty-one. The preponderance of females, therefore, was greatest before eight years. While the number of males cases was virtually the same each year from nine to fourteen, there was a sudden decrease in the number of female cases between eleven and

* Read by title.

twelve. The greatest number occurred at nine, ten, and eleven years, the total for that period being fifty-four, or forty per cent. As the actual date of onset in most of these cases preceded by several months at least the detection of the murmur, these statistics, as well as those of other observers with which they closely agree, would seem to point to the interval between the fifth and twelfth years as the period of greatest danger as regards heart-disease.

The family history regarding rheumatism was obtained in seventy-four cases with the following results:

Father	27 cases,	alone in	17 cases.
Mother	21 "	" "	11 "
Both father and mother			10 "
Brothers or sisters	12,	parents not rheumatic	4 "
Grandparents	25, "	" "	11 "
			53 "
No history of rheumatism			21 "

In seventy-two per cent., therefore, there was a definite family history of rheumatism, or, excluding grandparents, fifty-seven per cent.

In studying the causes of cardiac disease, the possibility of error is very great. Rheumatism is so uncertain in its manifestations in the young that its presence or absence is only to be ascertained by the utmost care. In obtaining a child's history, it seems almost impossible to always arrive at the exact truth. A mild rheumatic attack may have been unnoticed or its occurrence is soon forgotten. I have been impressed with the considerable number of children who have developed rheumatism in whom no rheumatic history could be obtained at the first visit. There is certainly ground for suspicion that in some of these cases there have been previous attacks. The following statistics have been, in all stages, prepared with great care, and I believe are as accurate as such figures can be made.

TABLE

showing history obtained at first visit in one hundred and seventeen cases.

Acute rheumatism	58 cases.
Joint pains or mild subacute rheumatism	25 "
Growing pains without other symptoms	8 "
Total, rheumatic	86 "

Chorea, without growing pains, joint pains, or rheumatism	7 cases.
Measles, without rheumatism or scarlet fever	9 "
Scarlet fever, without rheumatism or measles	5 "
Measles and scarlet fever, without rheumatism	4 "
No previous illness	6 "
Total, non-rheumatic	31 "

Diphtheria is so frequently confused with tonsillitis and the angina of scarlatina that it has not been considered. The total number of cases giving a history of measles was twenty-three, of scarlet fever, twenty-eight. The part played by these two diseases in the production of the cardiac disease was in most instances very uncertain.

Among these thirty-one apparent non-rheumatic patients there appeared, while under observation, rheumatism in eight cases, recurrent tonsillitis in two cases, and chorea without evidence of rheumatism in one case. At the first visit, therefore, eighty-six cases (73.5 per cent.) gave a rheumatic history. Adding to these the cases of rheumatism and tonsillitis developing later, the percentage is raised to 82.1. Erythema, fibrous nodules, and tonsillitis occurred in many cases, but, unless other evidence was also present, they were not classed as rheumatic.

The following intercurrent diseases appeared while these cases were under observation:

Rheumatism	23,	no previous attack in			8
Tonsillitis	9,	"	"	"	" 2
Torticollis	2,	"	"	"	" 2
Purpura	1,	"	"	"	" 1
Keratitis	2,	"	"	"	" 2
Chorea	11,	"	"	"	" 2
Pneumonia	3,	"	"	"	" 3

A history of chorea was obtained in thirty-nine cases, of which thirty-two were distinctly rheumatic. As an intercurrent disease it appeared for the first time in two cases, of which one was rheumatic. It was seen therefore in forty-one cases, or 30.3 per cent., being associated with rheumatism in thirty-three cases. These numbers, it will be observed, are not for all cases of chorea, but for those only with cardiac

lesions. It is a well-known fact that the murmur appearing during chorea sometimes disappears as the choreic movements subside. That occurred in several of these cases. It is in some instances, however, apparent rather than real. In two cases the murmur disappeared as the chorea subsided, but a few weeks later examination showed a distinct mitral regurgitant murmur. In other cases the murmur decreased in intensity, but soon increased and remained as a permanent murmur. Such a condition is probably due to the fresh lighting up of a valvulitis. An attack of rheumatism, perhaps very mild, might readily cause a fresh inflammation on an already sensitive endocardium. While I am strongly inclined to believe that the endocarditis of chorea is in fact rheumatic, in eight cases no history of rheumatism could be obtained, and no evidence of that disease appeared while under observation.

One patient, a girl of three years, gave no history whatever of rheumatism, chorea, measles, scarlet fever, or diphtheria, neither were the parents rheumatic. A murmur developed, or at least increased very markedly, during the course of a severe pneumonia, and remained as a loud, harsh, mitral regurgitant. In another instance, that of a girl nine years of age, there was a family history of rheumatism, but the patient had never shown any evidence of rheumatism and had not had scarlet fever nor diphtheria. During a mild attack of torticollis a blowing mitral murmur developed. This was unquestionably rheumatism.

The clinical history of heart-disease naturally divides itself into three periods,—that of acute inflammation, of compensation, and of heart-failure. For its intelligent management it is necessary that these distinctions should be kept clearly in mind. If the compensation is perfect, as it often is in children to a surprising degree, there will be no symptoms of heart-disease and nothing to treat. All that can be accomplished in any case not suffering from acute inflammation is to establish compensation. If that is already accomplished, it is the height of impropriety to treat the patient for heart-disease. The error must not be made upon the other extreme, however, that the physician has no duty in the case. Such children should be kept under observation both by parents and physician, lest

the condition of compensation be unexpectedly changed to that of heart-failure. Nutrition should be maintained at the highest possible point by diet and properly regulated out-door exercise, and sometimes by the aid of medicine. The child should be especially guarded against exposure to the exanthematous diseases, and above all else should be protected from conditions which may tend to precipitate an attack of rheumatism. Anæmia is a condition full of peril in heart-disease, for, when it is extreme, compensation cannot long be maintained. Yet it is exceedingly common, and is no doubt in many cases a result of the cardiac disease. Record was made upon this point in but seventy-one cases. It was present to a greater or less degree in fifty-nine, in sixteen being noted as extreme.

By far the most common cardiac symptoms were dyspnœa and palpitation. Palpitation was noted in forty-two cases, usually appearing only on exertion. Dyspnœa was but little less common, appearing in thirty-nine cases, in over half being noticed only on exertion. These symptoms were more marked when the aortic valves were at fault. Pain as an urgent symptom appeared in but eleven cases, and was most commonly associated with a mitral obstructive murmur. Cyanosis and œdema, in cases not suffering from acute endocarditis, were extremely rare. Epistaxis was not uncommon, but could hardly be designated as a symptom of heart-disease. The condition so frequently seen in the adult, marked by dyspnœa so extreme as to prevent sleep, tumultuous palpitation, and extreme cardiac distress, cyanosis, and dropsy, is a rare sight in a child under twelve years.

Pitt has drawn attention to a dilatation of the heart which occurs at about the age of fourteen years, even when no valvular lesion is present. It is characterized by anæmia, loss of energy, palpitation, and dyspnœa. The first sound of the heart is faint, and the pulse is weak, rapid, and irregular. This is explained by the great changes which take place in the heart and vessels at this time, especially in girls. From the seventh to the fourteenth year the heart increases very little in actual volume. There is then a period of sudden growth, when the heart increases from eighty to one hundred per cent.,

sometimes within the space of a year or two. This is, therefore, a most critical period and one which requires judicious management. I have in a number of instances seen a cardiac patient progress most satisfactorily until this age was reached, when the whole aspect of the case was changed. Compensation became imperfect, the child grew anæmic and gave the strongest evidence of imperfect nutrition. Development was retarded, though there might be growth in height, the child being thin and without strength or vigor. Sometimes he would develop into a fairly healthy youth, but in other cases, going from bad to worse, would finally succumb. In one case, notably, growth and development seemed to wholly stop at fourteen, and for three years there was a slow and steady retrogression until the girl died. Fortunately, many cases, probably the majority, pass safely through this period, often without perceptible inconvenience. Such patients, as a rule, develop into healthy men and women, and may never show symptoms of cardiac disease.

That endocarditis is sometimes an exceedingly obscure disease was proved by a number of these cases. It sometimes occurs without appreciable symptoms. When accompanying a rheumatic attack there is usually an increase in temperature, or slight fever appears if none has previously been present. The child seems more ill, more so, perhaps, than the arthritis would account for. There may be a peculiar restless, anxious expression with a tendency to cyanosis. The heart's action is disturbed with, perhaps, præcordial distress. The pulse becomes very rapid, this being one of the most constant symptoms. Usually a murmur quickly develops, though some observers have noticed that this sign may be delayed. In the great majority of cases it is mitral regurgitant in character. Sometimes there is a marked reduplication of the second sound heard at the apex, not at the base. This, when present, is very strong evidence of endocardial inflammation. I can confirm the statement that this reduplication is likely to be followed by mitral stenosis. I have seen but one death resulting directly from acute endocarditis. In that case the patient, a girl of nine years, five days after all rheumatic symptoms had disappeared, was not only permitted to leave

her bed against orders, but to go into the street, where she died suddenly.

In preparing this paper all doubtful cases have been rejected. One well-known author has included in his statistics "cases of thick sounds, thumping action, displaced heaving impulse." Such cases have been rejected, as the pathological conditions present are, to say the least, very problematical. The following murmurs were heard:

Mitral regurgitation in 124 cases, alone in 93 cases.							
Mitral obstruction	"	16	"	"	"	4	"
Aortic regurgitation	"	9	"	"	"	0	"
Aortic obstruction	"	26	"	"	"	3	"
Double mitral and double aortic.....					"	2	"
Mitral regurgitation and double aortic					"	3	"
Mitral regurgitation and aortic obstruction.....					"	18	"
Double aortic (alone).....					"	1	"

In four cases murmurs were heard, but were not differentiated. At the first examination four patients were suffering from acute primary endocarditis. Of these one died, two developed a mitral regurgitant murmur, and one a double mitral murmur. The statement that a soft blowing murmur is recent, while a loud harsh or musical murmur is old, is usually quite true; but it is a rule subject to many exceptions.

The following peculiarities in physical signs were noted:

1. The apex lies higher and more to the left than in the adult, being outside the nipple-line until four years and rarely inside until eight years.

2. The area of dulness is comparatively large, so that the normal heart may, without caution, be considered as hypertrophied.

3. The cardiac impulse is more clearly visible and can be felt more distinctly than in the adult.

4. Murmurs are heard over a comparatively wide area, especially the mitral regurgitant, which is not infrequently heard over the whole chest.

5. The rate may be readily increased and the rhythm disturbed by slight causes, so that in the examination of a nervous child rapidity and irregularity must be considered of very little importance.

One hundred and twenty-four patients presented clinical evidence of mitral regurgitation. If, as Sansom alleges, in pericarditis a murmur may be heard indistinguishable from the systolic murmur of mitral regurgitation, these were, perhaps, not all due to insufficiency of the mitral valves. The great frequency of the mitral murmur is beyond doubt, by this term being meant a systolic murmur heard with greatest intensity at the apex and conveyed to the left, either into the axilla or to the back. Such a murmur occurred in 91.7 per cent. of these cases.

The murmur of most interest or certainty, the one that has been the subject of most discussion, is the presystolic mitral. In one hundred and one cases it was noted sixteen times, being in relation to the mitral regurgitant as 1 to 7.75. Rheumatism as a factor in its production was less strongly marked than in either of the other murmurs. In four cases no history whatever of rheumatism or chorea was obtained in patient or family. Definite rheumatic history was obtained in seven cases, consisting simply of growing pains in two. In one case chorea developed later and in one acute rheumatism. Fifty-six per cent., therefore, gave a personal rheumatic history, the percentage for all cases being eighty-two. In the remaining cases the presence of rheumatism was somewhat uncertain. These facts bear out the statement of Sansom, that mitral stenosis is intimately associated with rheumatism, but most frequently with insidious varieties. The same fact has been observed by Chapin.

Symptoms, as in all varieties of valvular disease, were often obscure and at times entirely absent. They were, however, more marked than in simple mitral regurgitation. Pain was more common than with any other lesion, and dyspnœa on exertion was the rule. Palpitation was also common, while bronchitis and cough was more constant than in any other form of valvular disease.

It is universally agreed that the presystolic murmur is rare in infancy. The youngest of these patients was five years old; eleven were over eight years, six being between eight and ten. As far as I have observed, it is slow in its development, never appearing suddenly as does the mitral regurgitant. The

character of the abnormal sound is subject to change from time to time, more so than any other murmur. It may become very faint or even imperceptible, but I have not seen it disappear permanently. It is very sure to return, and hopes based on its disappearance are almost certain to be disappointed. There is frequently no perceptible cause for this changeability. Tonic treatment seems to have but little effect in causing permanent disappearance of the murmur.

The mitral obstructive is probably more frequently overlooked than any other murmur, yet it is quite distinct and characteristic. It is, as a rule, harsh and of a rattling, blubbering or wheezing character. It differs decidedly from other murmurs in one particular. Instead of rising to a maximum and then gradually decreasing or shading off into silence, it rises rapidly to the maximum and suddenly ceases as the apex strikes the chest-wall. Its area of diffusion is, also, very limited. As the stethoscope is carried from the apex, a point is quickly reached at which the murmur suddenly and completely ceases. If a regurgitant murmur is also present and the heart is acting rapidly, the two murmurs may run so closely together as to be with difficulty separated. In that case the first portion, or obstructive murmur, suddenly ceases at a given point, while the regurgitant remains unchanged. If the second sound is reduplicated at the apex, the certainty of mitral stenosis is increased. A thrill is by no means as common as in the adult. It is sometimes absent in well-marked cases. It was noted in but seven cases, but may have occurred in other instances.

One case, that of a girl of eight years, is of interest, in showing the order of development of the murmurs. When first seen there was a mitral regurgitant murmur, dating, no doubt, from an attack of diphtheria, which had been followed by renal symptoms. Two years later subacute rheumatism developed, and during the next two years was followed by several well-marked attacks with nodules. Four years after the first examination a mitral obstructive murmur was heard, and a few months later a thrill appeared, followed the next year by an aortic obstructive murmur, and this in turn by an aortic regurgitation.

Another patient, a boy six years old, was brought to the Polyclinic in April last. There was no history whatever of rheumatism or chorea either in patient or family. There were slight joint pains and pneumonic consolidation was detected at the left base. The heart-action was tumultuous, but no abnormal sound could be positively detected. Four days later there was unquestioned endocarditis and distinct pericardial friction sounds. The acute symptoms disappeared during the next two weeks, a mitral regurgitant murmur being left. He was not seen again until August, when a mitral regurgitant murmur was heard, and also a loud blubbering mitral obstruction, accompanied by a slight thrill. The boy was in excellent physical condition, and was supposed by the parents to be in perfect health.

The aortic murmurs were much more definitely associated with rheumatic histories than were the mitral. In the twenty-six aortic cases the family history was taken in seventeen, in twelve of which it was definitely rheumatic. In fourteen there was a clear history of articular rheumatism, and in four of indefinite and growing pains. In but three could a positive negative history be obtained. Nine cases were choreic. Nearly seven cases in eight gave a personal history of chorea or rheumatism. Two of the non-rheumatic cases had had scarlet fever about one year before, from which it is quite possible the cardiac lesions resulted.

While symptoms were in many cases absent or very obscure, they were as a rule somewhat more distinctive than in mitral regurgitation. Anæmia was almost constantly present; dyspnœa was noted fifteen times and palpitation fourteen. Both symptoms were more continuous, depending less upon exertion than with the mitral lesions. Pain was prominent in but three cases not associated with mitral stenosis. Cases of double aortic murmur were invariably marked by palpitation and dyspnœa, at least on exertion. Dyspnœa was by far the most constant and definite symptom.

In but few instances did the murmur wholly disappear, though a change in character was not uncommon, a loud, harsh murmur becoming soft and blowing, and at times difficult of detection. The two murmurs that disappeared were

slow in doing so, two years being required in one case and three and a half in the other. In one patient, a girl of nine years, who gave a history of chorea and rheumatism, there was a mitral regurgitant and loud obstructive murmur. During a period of four years she had several attacks of subacute rheumatism, but at the same time the aortic obstructive murmur gradually disappeared, while an aortic regurgitant developed. For over two years there has been no rheumatism. The murmurs remain, but are unaccompanied by symptoms. The girl has grown large and strong, and is apparently well.

Study of these cases tends to show that if intercurrent attacks of rheumatism can be prevented, the progress of aortic disease is not especially worse than that of mitral. That aortic disease is, however, a matter of great seriousness cannot be doubted. It is the result of an extensive endocarditis, and is in a large majority of cases closely associated with rheumatism. Children suffering from this form of valvular disease are especially subject to recurrent attacks of rheumatism with accompanying endocarditis. Taking this into consideration, the prognosis of aortic disease is far worse than that of mitral disease. While it is possible that the murmur may disappear as the child develops, the probability of such a fortunate outcome is very small.

Seven cases gave evidence of congenital disease or malformation. Space forbids extended discussion of this subject, and the pathological conditions are so varied that classification of symptoms or physical signs is impossible. I simply subjoin, therefore, a brief history of each case. A point to me of especial interest has been with reference to bronchitis and its possible action, by obstructing pulmonary circulation, in causing continued patency of the fœtal openings.

Case I.—A. P., four months old. Gave no history of cyanosis or other cardiac symptoms. Has always been weak and puny and subject to bronchitis. Was suffering from pneumonia, from which he died the following day. At the autopsy nothing abnormal was found at either valvular orifice. The foramen ovale was open, being the size of a lead-pencil, with smooth, rounded margins. It was, however, valvular in char-

acter, and probably allowed very little regurgitation. The ductus arteriosus was also open, the aorta being slightly contracted between it and the heart.

CASE II.—J. A., two years old; feeble and poorly nourished; has never walked; had always been subject to bronchitis, and had been cyanotic from birth. A very loud systolic murmur was heard, being most intense at the left of the sternum at the juncture of the fourth rib, and transmitted towards the left shoulder. This case was seen by competent diagnosticians, who agreed in the diagnosis of pulmonary stenosis with open foramen ovale.

CASE III.—S. O., two years old. When first brought for treatment was suffering from broncho-pneumonia, from which she recovered. History of cyanosis doubtful. A harsh systolic murmur was heard over the whole left side of the chest anteriorly. The point of the maximum intensity was well above the apex, to the left of the sternum, the bruit being transmitted towards the left shoulder. The certainty as to its congenital nature was, perhaps, less in this than in any of the other cases.

CASE IV.—T. K., three months old. Cyanosis marked while crying or excited, little or none when quiet. Systolic murmur heard loudest at second or third space at left of sternum, audible over whole chest, but more distinct towards the left shoulder than at the apex. The child died at the age of four months. The heart was found at the autopsy to be normal in size. Immediately beneath the aortic valve and above the muscular substance of the ventricular septum was an opening one-fifth of an inch in diameter, which communicated with the right ventricle, the aortic valves and coronary arteries being normal. This opening entered the ventricle at a considerable distance from the pulmonary orifice, at which there was marked stenosis, the right ventricle being greatly hypertrophied. The pulmonary artery was of normal size, and the valve was somewhat thickened and adherent, probably sufficient to allow regurgitation during life. The foramen ovale and ductus arteriosus were closed. The inter-ventricular opening was chiefly peculiar from its position, which was unusually high.

CASE V.—J. B., eleven months old. No history of illness except bronchitis, to which she was very subject. Cyanosis had not been observed. There was a blowing, systolic murmur, most distinct at the third space left of the sternum, conveyed upward and outward, but audible over a large area. During two and a half years the child has grown naturally, and has had no illness, except measles and an occasional bronchitis, which is never severe, but very rebellious to treatment. The murmur remains unchanged.

CASE VI.—C. B., three years old. No history of rheumatism or chorea in patient or family. Patient had never been strong, but had had no illness except bronchitis. No cyanosis was noticed till child was several months old, when it gradually appeared, and had markedly increased during the past year. When quiet it was scarcely perceptible, but upon exertion it became extreme. A loud, harsh murmur was heard just before and with the first sound, the point of maximum intensity being above the apex near the sternum. It was conveyed upward and to the left, but was heard distinctly at the apex. After two years the cyanosis is not quite as marked, but the murmur is unchanged. The boy is growing, but shows marked clubbing of the finger-ends.

CASE VII.—A. B., ten months old. Child was puny; has had bronchitis much of the time, but not severely. Cyanosis upon crying was marked, and had been noticed since birth. A loud, blowing murmur, systolic and probably diastolic, was heard over the whole chest; of maximum intensity at the left of the sternum above the apex.

PERITYPHLITIS IN THE YOUNG.

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THOUGH this disease has been much written about and much discussed within the last few years, it continues to be a subject of absorbing interest, both because of the frequency of its occurrence in the young, and because of the difficulties involved in

the solution of technical questions relating to its management.

Every physician has at some time been confronted with the problem, whether he should rely solely upon medical treatment in these cases or whether he should assume the responsibility of operative interference. If the case die without operation, he is prone to reproach himself with the reflection, "the patient might have been rescued if he had been operated upon." If, on the other hand, the patient recover spontaneously, elated the physician will exclaim, "I am glad that an operation was not performed."

These thoughts portray vividly the uncertainty which may surround our conclusions in these cases, and will warrant a discussion both profitable and instructive. According to its anatomical seat or pathological state the disease may be designated typhlitis, perityphlitis, paratyphlitis, and appendicitis. But inasmuch as it is not always possible to readily differentiate these varieties, the generic term perityphlitis is to be commended for ordinary description.

Perityphlitis occurs more frequently than is generally supposed, for many cases are so mild in character that they do not require treatment. It has been estimated that one-third of all necropsies reveal lesions which indicate that the subject had been the victim of an appendicitis at some remote period, although no history of its occurrence had been elicited during life. It is met with twice as often in males as in females. Among children perityphlitis is a common affection. In boys the disease is apt to be less severe than in men, and with a tendency to improvement in all the symptoms. Dr. R. H. Fitz, of Boston, in a careful analysis of four hundred and sixty-four cases of the various forms of iliac regional disease, found that fifty per cent. of them were under twenty years of age.*

In very young children the disease is rare. The youngest case on record occurred in an infant seven weeks old, which was reported by Dr. Demne. The duration of the attack is not constant. In children it is usually shorter than in adult

* Amer. Jour. Med. Sciences, 1887.

life. In its clinical course the disease may be either resolving or suppurative. If the attack is to end in resolution, it will in the majority of instances continue from fourteen to twenty-one days. If, on the other hand, suppuration shall have supervened, an early operation naturally will shorten the attack. Furthermore, in such cases which seem to have resolved and to have apparently recovered, adhesions resulting from former inflammatory attacks, or encapsulated abscess, or fæcal matter temporarily confined in a segment of the bowel by adhesive inflammation, may be among the causes of a revival of the disease in consequence of some accident or some indiscretion of the patient. These recurrent attacks may prolong the duration of the illness beyond the limit of time ordinarily reached in the resolution of a primary attack. The late Willard Parker observed that while the formation of abscess was of more frequent occurrence in the adult, perforation and gangrene of the appendix were more commonly encountered in children. Sometimes when pus has formed it will make its way out through the abdominal walls. Dr. Bull, of New York City, has reported sixty-seven cases of abscess, of which twenty-eight burst through the abdominal parietes, and only eight into the peritoneal cavity.

The anatomical structure of the cæcum and appendix, as it obtains in children, readily predisposes these organs to inflammation. Congenital or acquired irregularities in position and attachment of the appendix may favor attacks of inflammation. The narrow lumen of the colon and the tortuous character of the appendix render the latter especially liable to become the seat of inflammatory action. For while in the adult the ratio of the diameter of the colon to that of the appendicular process is as eight to one, in the new-born infant this ratio is only as four to one. The entrance of foreign substances into the vermiform process is also encouraged by this anatomical relationship. The greater irritability and proneness to inflammation of the intestinal tract in the young is another factor to be considered in the causation of the disease and of its frequency in early life. In children, as in adults, the same general causes, exclusive of abdominal tumors, will produce an attack of the disease. Among these may be enumer-

ated external violence, as falls, blows, efforts at lifting heavy weights, and so on. These usually act as exciting causes, for some internal condition is nearly always the predisposing cause. Most frequently, however, the attack depends upon internal causes alone. Among these are constipation and the presence of indigestible food or other foreign bodies. The irritation arising from the presence of lumbricoid worms has sufficed to bring on an attack. Reflex causes, like cold, have been assigned as the origins of an attack. And by some physicians the strumous diathesis even has been assumed to act as a predisposing cause.

Pathologically, the lesions in children are similar to those in the adult. The inflammation may have its starting-point in either the cæcum or the appendix, or in both. Thence it may extend to the peritoneum, or to the peritoneum and cellular tissue of the iliac fossa, constituting a complicated process of disease to which we have applied the generic term perityphlitis. In the majority of these cases it is more than probable that the peritonitis is secondary. Most cases take their origin in the vermiform appendix. Post-mortem examinations have disclosed the fact that the walls of the cæcum and the post-cæcal cellular tissue are in but few instances the seat of decided inflammation, and that then it is a result of the presence of impacted fæces or foreign bodies. Osler,* however, has observed perforation from round ulcer as well as other lesions in the cæcum. Abscess may form in the right iliac fossa, in the right lumbar region behind the cæcum, or in the pelvis. At times, when the acute symptoms have subsided, hard fæcal matter, intestinal concretions, gall stones, or other foreign substances, may be discharged from the body. In such cases it must be assumed that typhlitis or perityphlitis has occurred, the hypothesis that ulceration and perforation of the appendix have occurred being untenable. Although pathological confirmation is lacking to this assumption it is generally adopted, because no more plausible explanation has been offered.

It does not admit of doubt that the most common cause of

* Medical News, 1887.

perforation and ulceration is the presence of an enterolith or other foreign matter. In a number of cases, however, in which no foreign body could be discovered, it was evident that a simple catarrhal inflammation had advanced to ulceration and perforation. What the lesion may have been in cases which end in resolution and restoration to health is a matter for conjecture. In quite a number of such cases it might be fair to assume that the cæcum has been the seat of a simple inflammation, the resulting effusion forming the tumor which easily submits to the process of resolution. Numerous cases of this description have been reported. In fatal cases of appendicitis pus is undoubtedly present, with ulceration and perforation. Great importance attaches to the location of the deposits of pus in these cases. The collections of pus are frequently intraperitoneal, imprisoned in circumscribed sacs of peritoneal tissue produced by adhesion of adjacent loops of the intestines. Osler has said that a circumscribed peritonitis, moderate in degree, lasting for weeks or months, may follow a perforation of the appendix. This localized peritonitis of mild intensity may suddenly be converted into an acute peritonitis by the bursting of the small restricted abscess into the general peritoneal cavity.

In children a prodromal stage, lasting weeks or even months, often exists before a conclusive diagnosis can be established. Though diarrhœa is sometimes present, generally the patient is troubled with constipation before and during the illness. Vomiting is often a prominent symptom. Recurrent pains and localized tenderness over the region of the cæcum are the most constant and reliable symptoms. Yet pain is not uniformly present in the right iliac region, for it has been complained of in the epigastrium and even in the left iliac fossa. Pain is also sometimes felt along the course of the transverse colon. Walking and standing are interfered with. The right side is apt to be favored by the patient. The right leg tingles and is painful. At times that extremity is bereft even of its physical powers. The dorsal decubitus, with flexion of the right thigh, is the usual posture of the sufferer. The general febrile symptoms of inflammation are of course present. If within a few days the patient's temperature should become normal and

the local symptoms improve, the attack will probably end in resolution. If, on the contrary, the fever should continue, accompanied by an aggravation of the local symptoms, emaciation, feebleness, rigors, colliquative sweats, and profound anæmia, suppuration is undoubtedly proceeding. A point to be recollected, however, is that occasionally the formation of pus may take place with the absence of high temperature, and minus other *appreciable* clinical signs of the suppurative process.

Parotitis has been met with as a complication of perityphlitis. Local thrombosis and phlebitis, also phlebitis of the portal vein and its radicles with embolic hepatitis, have been encountered as complications of the disease. In several cases of perforated appendix pain in the genitals has been noted.

A history of prolonged constipation, with the development of febrile movement, protracted pain, and a tumor located in the lumbar region, which has a boggy feeling when pressed upon, denote the presence of a fæcal typhlitis. Acute intestinal obstruction is characterized by constipation, the early appearance of meteorism, the retention of flatus, and the late appearance of fever. Peripheral pain along the right lower extremity is apt to be misleading, for this disease sometimes stimulates coxitis. Dr. V. P. Gibney has observed eight or ten cases of perityphlitis in children who exhibited the characteristic deformity of hip-joint disease. Typhoid fever is to be differentiated by its clinical history and the absence of rigidity and tumefaction in the ileo-cæcal region. Intussusception should be discriminated from this disease by its bloody discharges and pronounced tenesmus. Impaction of fæces can be diagnosticated by a history of previous constipation, and by the presence along the course of the colon of an oblong swelling, with but slight tenderness and of a doughy consistency. In female children an oöphoritis as a sequela to gonorrhœal infection may be confounded with a perityphlitis. A physical examination of the girl and the presence of the usual vaginal discharge will enable the physician to make the correct diagnosis in such cases. The disease is also to be differentiated from a simple, deep-seated, mural abscess occurring in the

right iliac region, an example of which came under my observation within the last year. In these cases the physical signs of the various affections must be kept clearly in mind by the physician, for upon a proper construction of their significance must depend his diagnosis. Therefore abdominal pain more or less intense, tenderness on pressure about two inches from the anterior superior spinous process of the crest of the right ilium in a line between it and the navel, fever of a higher or lower degree, rigidity of the abdominal muscles, tympanites of varying amount, a tumor of greater or less size, and pain when the right thigh is extended, present to us a graphic clinical picture indicative of the presence of perityphlitis.

Although this combination of symptoms will render the diagnosis of the disease quite easy, the solution of the questions as to what stage the disease may have reached, as to whether pus has formed or not, as to whether perforation has happened or not, or even as to whether a general peritonitis may be present or not, will always be surrounded with difficulty. Suppurative perityphlitis may be concomitant with either a spreading or a limited peritonitis. Both of these forms of peritonitis begin with the same complex of symptoms, and it is not feasible to discriminate between them for the first twenty-four to seventy-two hours. The presence of any of the local signs of a general peritonitis will of course justify the diagnosis of a progressive peritonitis. Absence of such symptoms or their strict delimitation to the right iliac fossa will indicate a circumscribed peritonitis. In perforative circumscribed peritonitis a tumor will show itself in from two to five days. The difficulties of diagnosis are increased in these cases, because even when a higher grade of inflammation exists from perforation, there is sometimes no local pain over the seat of the lesion. The temperature record, too, in cases of perforation, is often fallacious, for it is not of infrequent occurrence among such cases to find the temperature subnormal. I can recall at least one case illustrative of this observation. In this case ulceration of a colloid cancer of the cæcum terminated in perforation of that organ. No clinical evidence of a peritonitis could be obtained, but when the autopsy was performed sixty-four hours after the probable time of perforation and six-

teen hours after death, the characteristic appearances of a peritonitis of many hours' duration stood revealed. Again, the classical signs of suppuration cannot always be observed. There is at times a marked disproportion between the symptoms indicative of suppuration, and the actual operation of that process in the person of the patient. I have seen at least two patients in whom pus had formed who lacked the usual symptoms of suppuration.

The relative anatomical position of the cæcum and appendix with their peritoneal attachments is favorable to the appearance of the tumefaction in the right iliac fossa, when the former organ happens to be the seat of the lesion. Hence when the swelling is not evident by palpation in this region, and the lesion is then suspected to be located in the appendix, it is very important to examine per rectum. Dr. Wm. Pepper, of Philadelphia, lays great stress upon the necessity of making such examination early in the history of the case, in order to determine the existence of inflammatory swelling and pus. In the differential diagnosis between retroperitoneal and intraperitoneal abscess, and between appendicitis and perityphlitis, many difficulties are involved. A sudden outburst of the symptoms of a localized peritonitis points decidedly to the occurrence of a perforation of the appendix. Inflammation of the cæcum and the cellular tissue behind it is *slower* in development.

General peritonitis resulting from a perforation of the appendix differs from a general peritonitis resulting from perforation elsewhere in this, that, in the first instance, the pain and tenderness begin in the right iliac fossa, that the inflammation is of more gradual extension, and that collapse is slower in its course. In order to determine whether pus be present, aspiration has been resorted to. Dr. McBurney, who does not look with favor upon the use of the exploring syringe, and in the beginning of the illness he unqualifiedly condemns its use, in which, doubtless, we all coincide. But it must be conceded that the risks are diminished if the procedure be reserved for cases in which the symptoms have lasted several days, and in which a distinct indurated tumefaction can be defined. In one of my cases in which it was doubtful whether pus were present,

inasmuch as the signs of suppuration were absent, aspiration was resorted to, and revealed its presence. The aspirating needle should be inserted obliquely, for when it is done so, the contents of the tumor are less apt to escape into the free peritoneal cavity and light up a general peritonitis. Dr. W. Gill Wylie, in a discussion on this subject at a meeting of the Northwestern Medical and Surgical Society of New York City, alluded to a number of cases occurring in Bellevue Hospital, in which such a catastrophe resulted from inserting the aspiratory needle in a vertical direction. We have also been warned of the risk involved in the insertion of the exploratory needle into an inflamed and ulcerated cæcum, lest a suppuration be induced thereby, which otherwise might not have developed. This, I imagine, is not so very likely to occur.

Not so very long ago, the rate of mortality of this disease among the young was estimated to be as high as seventy per cent., and that forty-four per cent. died within the first seventy-two hours. Greater precision in diagnosis and a more rational management of the disease have enabled us to greatly reduce this high rate of mortality. Ordinarily, when the disease is restricted to the cæcum and its surrounding tissue, the attack under the approved medical treatment will most likely be conquered. But unless care be exercised until all tenderness about the affected region shall have positively disappeared, relapses may be looked for. Dr. McBurney denies that the symptoms of the disease are in direct ratio to the gravity of the disease in a given case, and therefore deprecates the advice given by some writers to depend upon such relationship. It has been aptly said by Dr. Fagge, "that the course of the disease depends very largely upon the plan of treatment which may be adopted, for when the case is skilfully treated recovery may be anticipated." Though this be so, we must yet be guarded in our prognostications, for complications may unexpectedly arise at any moment to vitiate the promising outlook.

As regards treatment, the cognate affections—typhlitis, perityphlitis, paratyphlitis, and appendicitis—may all be considered as varieties of a localized peritonitis originating in the neighborhood of the cæcum. Therapeutically considered, we

may follow Treves in his division as follows: First, there is the mild form, which is amenable to simple medical treatment, and usually ends in resolution. Secondly, a form which ends in suppuration nearly always depending upon some trouble originating in the appendix, and in which a prompt and definite surgical interference is indicated. Thirdly, the relapsing type of appendicitis, in which operation should be done only after the inflammatory symptoms have subsided.

In the treatment great efforts should be made to secure if possible a termination of the attack in resolution. Absolute rest should always be enjoined in the treatment of a case of this disease. In the young, particularly in those instances in which colicky and obscure pains are experienced at varying intervals, the least tenderness and pain in the right iliac region should be viewed with suspicion, and should prompt us to enforce the principle of treatment just mentioned in conjunction with a proper kind of diet. For in these insidious cases among children, perforation has been known to result after the administration of a laxative, or the use of enemata.

A very important thing in the dietetic regimen is the undesirability of letting milk form the staple article of diet in these cases. The milk is to be discarded or at least curtailed, because the hard curds formed by the coagulation of the caseine will act as irritants to the inflamed tissues. Hence meat juices and extracts, beef-tea, chicken, veal, and mutton broths, are to be preferred in the nourishment of the patient. Unusual obesity of the patient rather dictates an expectant mode of treatment. Usually the patient has taken a cathartic before the physician is summoned. If not, a mild laxative may be administered in the beginning of the attack. Later cathartics and enemata must be withheld. To relieve the pain opium and its alkaloids, morphine or codeine, which latter is preferred by many, should be prescribed. Local depletion, counter-irritation, and warm fomentations are employed at the same time. In young children, Silberman prefers the use of chloral hydrate to that of opium. In a case of simple typhlitis due to fæcal retention, saline cathartics in moderate doses with an ample quantity of water will be serviceable. The employment of leeches in the management of these

cases has received considerable attention. It has been recommended to apply from four to eight of them where the pain is most prominent. Sinapisms, tincture of iodine, and blisters have been used as counter-irritants, particularly if the tumor appears slowly.

In reference to the use of cold or warm local applications no uniformity of opinion seems to prevail. The broad statement has even been made that the feelings of the patient should decide which should be employed. Though *a priori* cold would seem to be indicated, I prefer the warm applications. My reason is, that the warm fomentations would encourage the production of a plastic circumscribed peritonitis, so that in case pus should form it would become encapsulated, so to speak, shut off from the general peritoneal cavity, and less liable to burst into it. Cold, though it may be grateful to the patient, will hinder this process. For the support of the flexed thighs a pillow should be placed under the knees. In persistent vomiting, rectal feeding and stimulation may be employed. As local sorbefacients, belladonna, mercury, and iodide of potassium ointments have been suggested. I omitted to mention that one drawback to the use of warm poultices locally is the production by their use of a superficial œdema, which may obscure the local evidence, and thus interfere with the accuracy of diagnosis. To relieve the uncomfortable feeling of distention when excessive tympanites is present, rectal bougies should be used to lead it off. Puncture of the intestines to relieve this condition must be condemned in these cases as perilous on account of the paralyzed state of the intestinal walls.

Inasmuch as many patients who exhibit all the typical symptoms of the disease recover without resort to surgical measures, it must be acknowledged that as yet we have no unvarying rule to guide us in determining which cases do and which cases do not need operation. If after the first three or four days no abscess can be discovered by physical exploration, surgical interference is not to be thought of. If, however, the patient is growing daily worse, and there be evidence of pus after the third day, an exploratory operation should be performed before the end of the first week, or as soon as

the symptoms demand it. If pus be found, the operation should be carried on to its conclusion. In doubtful cases the risk of an exploratory operation is less than the risk involved in the evolution of an attack of the disease. Besides, tumefaction is not always distinct and not always perceptible, therefore, it cannot always be relied upon as a guide to operation. In acute, rapidly progressive cases, even if no pus can be discovered, an exploratory incision is justifiable. A fulminant peritonitis, due to ulceration and perforation of the appendix, calls for early operation, for delay here invites a fatal termination. In order that the operation may be successful here, it must be performed at the onset of urgent symptoms.

The results of early operation have been so favorable that so soon as a tumor, accompanied by the symptoms of suppuration, has formed, and it can be distinguished, it should be subjected to surgical treatment. Such eminent surgeons as McBurney, Bull, Weir, Abbe, Fitz, and Sims are in favor of early operation. It is claimed that early operation diminishes the risk of the extension of the inflammation, and the danger of rupture into the general peritoneal cavity. If a spreading and a threatened general peritonitis shall have been lighted up, then the major operation of laparotomy is indicated.

In his excellent paper,* read before the Practitioners' Society of New York City, Dr. R. F. Weir urged that, inasmuch as the mortality is greater before the third day when a perityphlitic abscess has resulted from a perforative appendicitis, the pus, so soon as it was recognized, should be evacuated, extraperitoneal, if possible, or else by a lateral laparotomy with drainage of the cavity. He also states that if aspiration fails to detect pus whenever a tumor can be discovered, then an extraperitoneal incision should be made as for laparotomy.

Absence of urgent symptoms or their strict localization will, in my opinion, warrant a delay of varying length. Here within a reasonable time, after the beginning of the disease and after exploration shall have revealed the presence of pus, the abscess should be evacuated by aspiration or by incision, which must reach the pus whether it be extra- or intraperito-

* New York Medical Journal, May 6, 1887.

neal. It will not be out of place to narrate briefly, in this connection, the history of a case of perityphlitis which occurred in my practice, in which the abscess was evacuated by aspiration, the patient making a perfect recovery.

CASE.—Harry F., aged five years, was suddenly seized with severe pain in right iliac region. He was treated in the manner indicated in the earlier part of this paper. Within four days a slight swelling was discerned on the seat of pain. Dr. Varick, of Jersey City, was asked by the family to see the patient with me. The doctor agreed in the diagnosis of perityphlitis with possible abscess, and we decided to aspirate in order to verify our surmises. We succeeded in obtaining pus with the exploratory syringe. On the following day the patient was anæsthetized, and with a Dieulafoy instrument about four ounces of fetid pus was removed. Nothing worthy of note occurred from that time on to his ultimate recovery.

Collapse and hyperpyrexia are contraindications to operation. For obvious reasons no purgative whatever should be given before operation. Great abdominal distention should, if possible, be relieved before operating. After an attack shall have passed by, the greatest care is necessary to prevent a relapse. No purgatives should be given, the bowels being moved by enemata only. For a short while after recovery, restriction to liquid diet should be enforced. The patient should not be permitted to get up too soon. Recurrences of the attack, which are so lamentably frequent, can often be prevented by regulation of diet, moderation in daily exercise, and securing regularity of stool. Tonics and stimulants should be administered to regulate the peristaltic action of the bowels and to improve the general health of the patient.

Children who have passed through an attack of this disease, and even those who frequently complain of abdominal pain, should always wear a tight bandage to keep the bowels from jolting. In conclusion, let me recapitulate in a formulated summary the treatment of the disease as suggested by our present knowledge of the subject.

First, in cases of a chronic or subacute perityphlitis, without a tumor or with a small tumor, the expectant plan of treatment may be followed.

Secondly, in cases of chronic or subacute perityphlitis, with a growing tumor with or without evidence of suppuration, extraperitoneal incision or aspiration should be done.

Thirdly, in cases of acute perityphlitis with threatening symptoms with tumor, and hyperacute cases without appreciable tumor, exploratory incision (extra- or intraperitoneal) should be made.

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DISCUSSION.

Dr. Putnam.—I have two slight points to speak of. One is in regard to the pain. The pain of a general peritonitis is likely to be more diffused than that caused by local peritonitis, but it is not always so. The pain is not localized at all at the beginning of one of these attacks. Localization is often made out by the physician before it is by the patient. I mention this merely because I have seen physicians led astray by not finding out that the absence of local pain did not mean that the disease was not local. As to food, one does not want to give up too readily the administration of milk; very much diluted milk, in small quantities, will give a child better nourishment than chicken broth, which is principally salt and water with a little fat. Chicken broth is slightly nutritious and gives fluid, but is not to be compared with milk.

SCORBUTUS IN INFANTS; AMERICAN CASES.

(ILLUSTRATED.)

BY WILLIAM P. NORTHRUP, M.D.,

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THE object of this paper is to present some cases of infantile scurvy occurring in this country, and to intimate something regarding their frequency of occurrence. As to the essential nature of the disease there is no positive recent contribution to literature, nor has the writer anything new to offer. After the valuable papers on scurvy in children to be found in *Keating's Cyclopædia*, and in that most admirable book of Eustace Smith's, there remains nothing to say until some investigator shall announce the long hidden secret of the essential causative factor which gives rise to the lesions which we, by common consent, denominate scurvy—be it in infants or adults.

The first case reported in this paper is so typical in lesion, course and symptomatology; in its ætiology so suggestive and in its treatment so successful; in short, so typical and instructive is the case, that it has seemed best to the writer to set it forth in detail, with the statement that it illustrates nearly every point mentioned in the two articles referred to above. As these volumes are probably in the library of every member of this section, it is

useless to duplicate words. If it shall appear that scurvy in children is not of frequent occurrence, there is yet a certain advantage in considering such cases, especially if they are liable to spring up in the path of the general practitioner at any turning.*

CASE I.—*Typical; scurvy occurring in rich surroundings; recovery; patient aged sixteen months.* In the midst of a pouring rain on a Sunday morning, a carriage drew up to the curb, the bell gave two quick jumps and in a few minutes a man was marching up and down the office floor. The cause of his nervousness was quickly announced. His second child, an infant, had rheumatism and had become no better during the fortnight's sojourn of himself and wife in a neighboring capital. The child in their absence had been in the care of an old nurse, so gifted in the art of nursing and, I may add, in self-conceit. On his return home the previous evening, the infant was not well. It was not very sick, so he thought, when he went away, and by this time he supposed it would have entirely recovered—instead, it was worse. He sent for the doctor and, in his anxiety, desired an immediate consultation. His family physician was among the ablest practitioners in New York; he had not been called to the child within a week; had not been informed by the nurse, if indeed he had not been misled by her. The history of many cases can be written backwards better than consecutively, and the present one is of this character. Suffice it to say, in preface, the diagnosis was rheumatism and the treat-

**Scurvy*, by Thos. Barlow. Cyclopædia of the Diseases of Children, Keating, vol. ii., page 277.

CONCLUSIONS—(CHARACTERISTICS.)

"(1). Predominance of lower-limb affection; (*a*) immobility; (*b*) excessive tenderness; (*c*) general swelling of lower limbs; (*d*) skin shiny and tense but seldom pitting and not characterized by undue local heat; (*e*) on subsidence, revealing a deep thickening of the shafts; (*f*) liability to fracture near epiphyses.

"(2) Swelling of the gums, varying from definite sponginess down to a vanishing point of minute transient ecchymosis. . . . .

But to them must be added as:

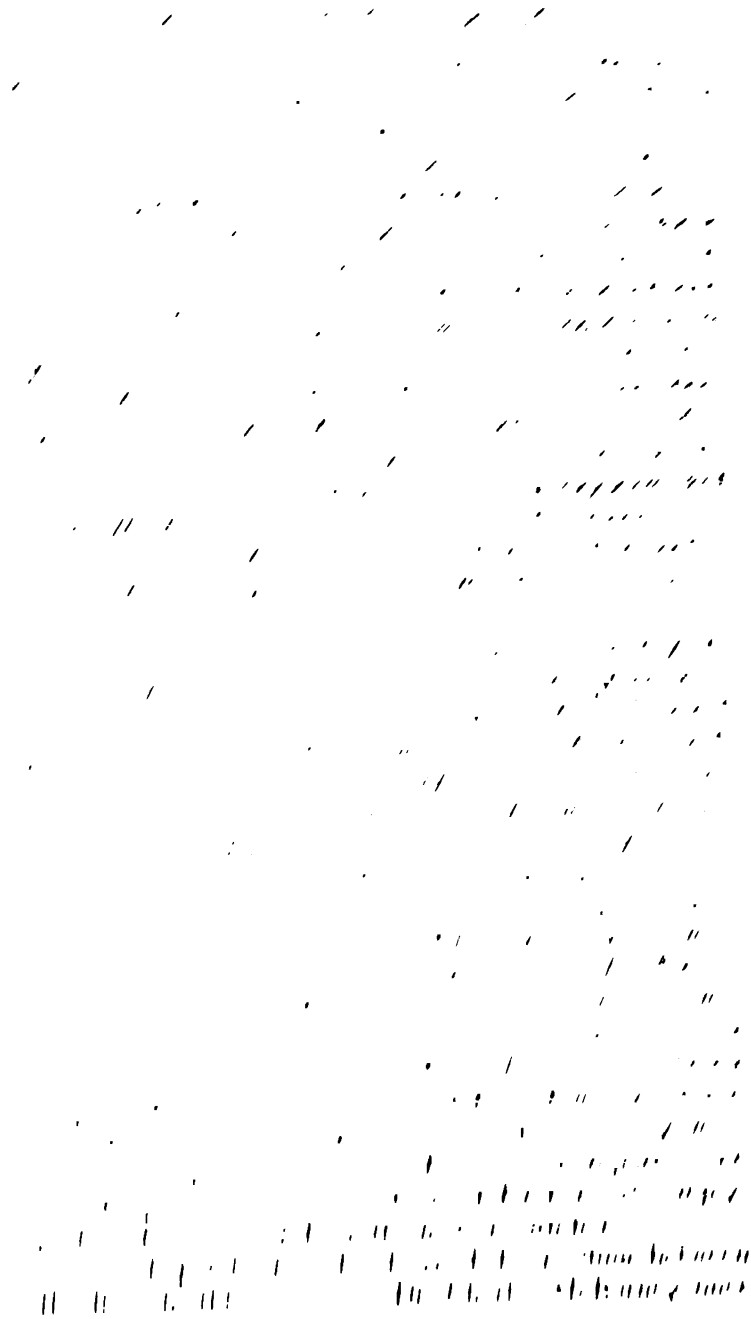
"(3). The most diagnostic of all, definite and rapid amelioration by antiscorbutic regimen."

ment had embraced salicylic acid and tincture of iron, without success.

At the time of the consultation the father and mother of the little patient were present; both were within the thirties, healthy and vigorous, the father looking like a hardy yachtsman. The family history on both sides was good. The family were luxuriantly housed in the most hygienic surroundings of up-town New York. In short, the infant, whose history we are going to consider, who was suffering from malnutrition, from "insufficient and poor food," to quote words so often used in connection with children's diseases, was the offspring of wedded representatives of two rich and powerful families in the fashionable and political world of the great metropolis. There was no lack of love and devotion on the part of parents or grandparents. The lack was in wisdom and judgment, the abundance, the nurse's self-esteem and ignorance; these allowed the present case and history to continue on to full accomplishment.

The child at this time was sixteen months old; was the second born; a female; thriving very well in the early months of life; at the fourth month the mother's milk failed to be of sufficient quantity and quickly, thereafter, ceased altogether. The first born is still living and robust; the mother's milk had after a few months been insufficient for it, but the child continued to thrive. After the failure of the mother's milk, in our patient's case, one of the proprietary foods was given her. By some misunderstanding this food was diluted with water and milk, the proportion of the latter being too small. For a time the child thrived very well, apparently, though it was rather backward about walking and talking. Its digestion was good, its bowels reasonably satisfactory and it seemed satisfied with its food. It never gave any evidence of rickets; teeth in usual quantity made their appearance at the usual time.

Three weeks before the visit spoken of (this fact was subsequently elicited after questioning both parties) the nurse had noticed some *change in the child's gums*,



After reflecting a week upon this she called the attention of the mother to the same. The change was not marked. While on a visit to another patient in the household, the doctor's attention was called to the child as he was hurrying out to his carriage. The baby was reported as not quite well and nothing was said concerning the gums. He hastily looked her over, learned of the misadventure with the proprietary food, added the requisite amount of milk to bring it up to the prescribed standard, ordered beef-broth, iron peptonate, and was gone.

One week later than this, the patient developed *trouble in the right lower limb*, evidenced by worrying, sensitiveness on handling and tendency to keep the limb nearly straight. Anti-rheumatic remedies were prescribed. There seemed no reason why the case should not speedily come out of its condition of slight depression, now that the food was improved and treatment instituted.

During the succeeding week very little is known concerning the child; the parents were absent from home; the family physician was not called; the nurse drew no conclusions from the, now rapidly-changing gums, and as to the rheumatism, the progress was slow. It is not difficult for physicians to understand how this infant, once started on its career of malnutrition, could even in the house of luxury, under these circumstances, develop ripe symptoms. At this juncture the parents returned home to find the rheumatism worse, instead of cured, and a hasty muster of physicians was the result.

The child cried on seeing a strange face, becoming alarmed also for the safety of its lame leg. In the wry face of crying, the little patient fairly *unbuttoned* from between its lips two rows of irregularly nodulated, purplish gums, from the summits of which the points of its teeth fairly protruded. *In the upper spongy row was a depression with ulcerated walls and sloughing shreds. The gums were dark and bled freely* in the act of crying, from compression of the lips alone. There was nothing further abnormal about the face beyond a worried expression; no ecchymoses; no

petechiæ; conjunctivæ were normal; no evidence of unhealthly condition of the mucous membrane of the nose. There was no history of nose-bleeding, no hæmaturia, no hæmorrages from the bowels. The child was now stripped of all clothing and laid on its back on the bed. It continued to whimper, throw its arms about freely, draw up its left leg; as for its right, it could move it slowly, but only a little, and could not be induced to flex it. The *right thigh was somewhat larger than the left*, to observation; by measurement it showed a difference of about two and a half inches, which, considering the thin thighs of the small patient, augured a marked difference. The enlargement was fusiform, greatest just above the knee. Apart from the *spongy gums and swollen thigh* there were no external manifestations.

The diagnosis of this case is scorbutus; what confirmation of this diagnosis can be offered? The answer is, the success in treatment.

The child was removed at once to the country, its proprietary mixture was stopped and in its place were given fresh cow's milk, expressed juice of beef, baked potatoes, also citrate of iron with excess of citric acid. The one thing which this scurvy case seemed to crave, for which it reached out, which it seized with ravenous avidity, was *orange*. The child could hardly be restrained till it held the fruit in its grasp; and then proceeded to souse its lips and nose in the juice. Improvement began at once; in five days its gums were markedly better, in ten entirely normal. As for its thigh, at the end of ten days the improvement was marked both in sensitiveness and size, and a month later the child was standing on its feet. A slight thickening over the femur could be detected for a few days, at the end of which time, the patient seemed absolutely well. "Now," to quote the words of the latest report, "she would strike you as a child small for her age simply, nothing having ever been the matter with her."

Several points may be learned by reading between the lines in this case. As Dr. Charles McBurney once

very aptly said, speaking of the value of original investigation: "There is also great value in *re*-search." At least we may argue, that in cases of spongy gums and pains in the knees it is well to consider the diagnosis of scurvy. A certain amount of sinister interest attaches to the fact that this child was vaccinated a month before the first symptoms appeared, and all medical men connected with the case were catechised as to the near or remote possibility of its causal relation to the lesion of the gums and thighs. It was, by the way, vaccinated on the leg. Needless to add, its course was perfectly typical for an effective inoculation and was followed by speedy healing.

To entirely complete the disease-picture given us by the English authorities, nothing is required but the post-mortem findings, which the following cases furnish.

CASE II.—*Fatal scurvy in a child of eighteen months; Autopsy.**

This child was an inmate of the New York Foundling Hospital and was what is called a "nurse-baby," that is, she was nursed by a mother who, in addition to her own baby, nursed a second of about equal age. Her own child thrived; the second furnishes this example of malnutrition and the pathological specimen which is here shown. The nurse was said to be very fond of the child and "took on" when it sickened and died. Since we are considering a case of scurvy developing in a breast-fed (*sic*) child it is well to bear in mind the above facts and the added fact that nearly all babies nursing two at one woman require to be fed more or less. We are justified in forming our own conclusions as to which was fed more and which less; we know which baby was hers and which was not, which thrived and which developed fatal scurvy; we also have our notions of the tendencies of maternal instincts. However, the history must be recorded as

* Reported in Proceedings of the N. Y. Pathological Society, 1889, pp. 66–67.

those in charge of the case furnish it, and it reads, "the child was wet-nursed the first sixteen months of its life."

Briefly the history of the illness was as follows: This female child when sixteen months old was observed to be failing, and as the history reads, "on account of impaired nutrition was taken from the breast and was given vegetable acids." In the seventeenth month of life, which was one month before death, the right leg and knee became swollen and tender. Temperature was 101° F. After two days the symptoms seemed temporarily to disappear. Two weeks before death and six weeks since the weaning, the child appeared to be very sick, her gums were swollen, smoky-black and bled freely; two days later her left eyelid became swollen, black, having the appearance of the classical "black eye." Temperature thus far continued about 101° F. One week later there developed the physical and rational signs of pneumonia. At this time her other eyelid became ecchymotic and the other thigh markedly swollen.

During the remaining days of life the small patient became excessively anæmic, having a metallic pallor, which gave a particularly wretched appearance with the contrasting ecchymoses about the eyes. Her passages were black and pasty; no petechiæ; the child failed rapidly and died with pronounced symptoms of pneumonia.

Autopsy.—The main interest lies in the condition of the legs. As regards the organs, it is sufficient to say there were no hæmorrhages; extensive pleuro-pneumonia of left side. Left thigh symmetrically enlarged, larger than the right, though both were obviously above normal in size. Left femur was, at its upper extremity, normal, epiphysis and end of shaft. The lower half was invested about with a black grumous subperiosteal layer of blood having a thickness of two to three millimeters. The lower epiphysis was detached, the lower end of the shaft macerated, eroded and soft, lying loose in the

black disintegrating blood-clot. The femur of the companion leg was surrounded for its lower two-thirds by a thinner, black, subperiosteal blood-layer—(See illustration).

The lower epiphysis was not detached though both it and the shaft were congested. No hæmorrhage into any joints. The right and left tibiæ were surrounded by a thin, dark hæmorrhagic layer beneath the periosteum and the proximal portions of both were congested. The fibulæ, likewise the bones of the upper extremities, were normal.

The accompanying illustration is drawn from a specimen which consists of a lateral half of the lower limb of the side less affected.

Microscopical examination of the bone disclosed no syphilitic or rhachitic changes, and no inflammatory changes in bone or periosteum. The softened, macerated bone gave no evidence of suppuration; moderate congestion of the fellow femur and the upper extremities of the tibæ.

CASE III.—Dr. Richard Van Santvoord has reported* the autopsy of a helpless idiot, six years of age, in whom extensive hæmorrhage, with separation of both upper epiphyses, had occurred under the periosteum of the upper halves of both humeri. This hæmorrhage was apparently the immediate cause of death. The case was regarded by him at the time as probably traumatic. Reports that he had subsequently seen of extensive subperiosteal hæmorrhage, with epiphyseal separation, had been regarded by their recorders as scorbutic. He had become convinced that his own case belonged under that heading. Gums were not mentioned as being involved.

CASE IV.—Dr. August Caillé contributes for use in this paper, an *autopsy* record of a typical case of scurvy—autopsy by himself. He says:—"In addition to the external appearances there were subcutaneous effusions of

*Ibid.

[Drawn from Specimen by H. McDONALD, M.D.]

Specimen from a case of Infantile Scurvy, showing subperitoneal hæmorrhage about femur and tibia of the side less affected.

blood, hæmorrhagic spots on divers mucous and serous membranes, swellings at the joints and subperiosteal effusions of blood at the epiphyses."

The three last narrated cases furnish the only autopsies the writer has been able to collect. Bearing in mind the complete picture of disease furnished by the previous cases, let us consider the following ;

CASE V.—Dr. Chas. H. Richardson of the North Eastern Dispensary—children's class—has furnished the following most interesting and illustrative case.

Anne K., aged twelve months, was brought to the dispensary by its mother with the history of "failing" for nearly two months. The child was healthy for some time after birth, when the mother was obliged to work, and of necessity the baby was "farmed out." She reported the food as having been condensed milk, adding the somewhat ambiguous item, "and what was going."

The child had recently vomited blood, passed blood from the bowels and presented petechial spots of subcutaneous hæmorrhage. The gums were slightly softened and according to the mother's account the inside of the mouth had been black. Both legs were enlarged to considerably more than double their normal size. Epiphyseal ends showed marked enlargement. Limbs very tender, hard and pitted slightly on pressure."

"Head sweats. Temp., 100⅘°. Tendency to spinal curvature in dorsal region."

To continue the verbatim quotation of the doctor's letter :—"I considered this a combination of scurvy and rickets and put the child on a live diet of fresh milk, orange and lemon juice." Three days from the first observations and prescription the purpuric spots had mostly disappeared, and no new ones had occurred. The child seemed slightly better; there was no vomiting, yet the condition of the limbs was unchanged and the temperature continued slightly elevated as before.

The following paragraph gives the case an added interest and accuracy: "The swelling of the limb being due to

subperiosteal hæmorrhage, added to subcutaneous œdema, I wished to see the character of the effusion next the bone. I thereupon passed an aspirating needle down to the femur and drew off some of the effused fluid. It consisted apparently of pure blood, which microscopically showed the red globules, somewhat ragged and disintegrated, and the white ones somewhat increased in number."

At this point the case escaped from observation and the history ends.

CASE VI.—Dr. Hermann Goldenberg, at his class in the Mt. Sinai Hospital Dispensary, observed the following case of scurvy:

A boy, two years old, was brought in by his father, who stated that the patient had been well up to a few days previous, when he noticed red spots over all his body, swelling of the legs, bleeding from the mouth, bowels, nose; also hæmaturia. The food of the child consisted exclusively of milk.

The family history was good; a number of older children were strong and healthy; no malaria; no syphilis. To quote Dr. Goldenberg literally:—"I found a hæmorrhagic eruption in nodes, spots and large patches, up to the size of a palm of a hand, on face, trunk and extremities. The hæmorrhagic character was clearly demonstrated by the fact that the redness remained on pressure, and the changes in color, within a few days. The gums had the characteristic color and form, as in scurvy. There were gangrenous spots in pharynx and on tonsils, swelling of the feet and legs and of the scrotum, through extravasations into subcutaneous tissue. Fœtor of the mouth; urine which the father brought was bloody; general malaise. No signs of rickets." Dr. Goldenberg presented this case before the Mt. Sinai Alumni Association as a characteristic case of scurvy. It was accepted as such. One member temporarily dissented, preferring to call it hæmorrhagic urticaria. The patient was on the following day sent to Dr. Jacobi, who confirmed the diagnosis and pronounced the case scorbutus. Lemon juice, fresh vegetables, potassium chlorate, internally and

as gargle, were ordered and within a few days the child was well.

CASE VII.—The following case was narrated to the writer by the physician to the child, whose case was first narrated. The patient was a boy sixteen months old; the fifth child of a father, of high rank in the army, having a physique which the doctor fittingly typified as by the word "bull"; the mother was a delicate woman; its four or five brothers and sisters were all delicate. The child was well housed and cared for, or to use the words of the narrator, "over-cared for"; was wetnursed for a few days only. Thereafter, it was fed on a proprietary food. Up to the sixteenth month of life it seemed to be well nourished and attain good development. At last, its gums became spongy, then its right ankle became swollen, discolored, bluish, tender, fluctuating. This patient was seen by an able consultant, an honored member of this society, and pronounced rickets—probably meaning *acute rickets*.

On a treatment of lime, muriatic acid and tincture of iron the child slowly and gradually recovered. The symptoms in this case were spongy gums and hæmorrhages about one ankle. No mention is made of petechiæ.

CASE VIII.—Dr. Delafield furnishes the following interesting case. In the month of June, a child about three years old, suffering from obstinate diarrhœa, was brought to him for consultation. He prescribed an exclusive meat diet and advised the parents to take it at once into the country. They did as directed, and the child speedily recovered. So pleased were the parents with the prompt effect of the diet in the pronounced improvement of the patient that they believed they had learned a valuable secret for securing the continued health of the child.

They, without hesitation, settled down to a rigid *regime* of exclusive meat food. This they followed conscientiously for two or three months. In the last of September, the condition of the child induced them to return to town and again consult Dr. Delafield. This time the pa-

tient presented a characteristic case of scorbutus. To use the words of the narrator: "The diagnosis was perfectly clear; had characteristic signs of scurvy." On a rational diet all symptoms quickly disappeared and the child became quite well.

CASE IX.—A letter from Dr. L. Emmett Holt gives the following: "Some points regarding a scorbutus case; the first symptoms appeared in the latter part of April, the infant being at that time eleven months old, and having had all the symptoms of extreme marasmus for several months. From the latter part of February she had been upon an exclusive diet of a proprietary food. There was first noticed a swelling of left knee resembling in general appearance that due to articular ostitis; a week or more later the gums were spongy, purplish, and slight hæmorrhages occurred from time to time. The swelling about the knee increased steadily during the next six weeks until it was fully four inches in diameter; the swelling extended up to the middle of the thigh; there was extreme tenderness and later a slight purplish discoloration of the skin. Exploratory puncture gave blood only. The child died on June 23d. The above symptoms continued together with those of extreme anæmia and exhaustion; there was a slight fever for the last two months; at no time was there any ecchymoses beneath the skin. At the autopsy the swelling of the limb was found to be due to subperiosteal hæmorrhages, the bone being surrounded by a mass of old blood-clots; the periosteum was extensively stripped up; there was complete separation of the lower epiphysis of the femur.

CASE X.—Dr. W. F. Lockwood, of Baltimore, furnishes the following: we quote the letter in full, verbatim.

"Aug. 12th.—Mrs. W——, whose family had been in the country during the warm weather, asked me to visit her child, thirteen months old. She stated that it had been ailing for some weeks with diarrhœa varying in severity, but never entirely relieved. Of late she had thought the child suffered pain in its lower limbs, that it screamed in an unusual way at night, especially when moved or

touched, and that there was increased fretfulness during the day. She referred also to some spots 'like old bruises' on its legs below the knees, more marked on the right.

"It had been nursed until six months old, after which time it had taken almost exclusively a proprietary food.

"Examination showed the child fairly well nourished, rather anæmic, complexion sallow. No evidence of rickets, no swelling along shaft, or enlargement at ends of bones. Ill defined ecchymotic spot on left leg, but plainer on the right. The right leg semi-flexed and everted. No tenderness manifested on gentle manipulation of body or limbs. Gums showed dusky purplish fold at root of each tooth. They had bled occasionally for some weeks and the stool had pretty constantly been streaked with blood. Pulse and temperature were normal.

The diagnosis was made of scorbutus and directions were given to change the diet to fresh milk undiluted, potato and orange or peach juice. A few days after my visit the mother reported the child improved in every way. The diarrhœa had stopped and there was no screaming or restlessness at night. A second visit was not required and the improvement, I have heard, has continued."

CASE XI.—Dr. G. H. Whitcomb, of Greenwich, N. Y., has, since the writing of this paper, published a most illustrative case [*Archives of Pediatrics*, Oct. 1891, p. 760, of *Artificial Baby Food and Scorbutus*.] The child was eleven months old; after fifth month was fed exclusively on artificial food, during which time it grew very fat, "seemed to thrive famously." "When a little over ten months old she became petulant, and evinced a disinclination to move or be handled. The legs were partially flexed and remained rigid. Any attempt to straighten them elicited screams. The gums were spongy and bled frequently; muscular pains were so severe as to deprive the child of rest." "The physician at that time in charge diagnosticated and treated rheumatism. . . . After ten days the family went to Rome, . . . where the diagnosis of rheu-

matism was approved and alkaline treatment pursued for two weeks, after which they came to Greenwich."

In Dr. Whitcomb's graphic account occur such expressions as the following : " screamed when handled . . . limbs resembled Bologna sausages . . . gums were spongy, ecchymotic blebs discharging sero-sanguinolent fluid . . . seven teeth . . . *no evidence of rachitis*." Treatment comprised fresh milk, rare-broiled beefsteak and sweet oranges.

In three weeks the child was restored to complete health. The doctor believes the scorbutus to have been caused by the exclusive use of the prepared food and he concludes that "no cereal or chemically prepared food can nourish perfectly," and should be supplemented with fresh milk, meat and fruit juices.

During the summer just past, the writer has had opportunity in three great medical centres, personally, to compare notes on the subject of scorbutus in young children.

Prof. Ranke, of Munich, said that with him it was "in the highest degree of rare occurrence." He could not at once recall a case.

The assistant of Prof. Widerhofer in Vienna, Dr. Karl Foltanek, at that moment surrounded by a throng of wretched out-patients at the children's hospital, declared that in his experience, and it must be considered a colossal experience, "scorbut" in children was "ungeheuer selten."

Dr. John Abercrombie, of Great Ormond street Children's Hospital, in London, believed it not frequent, though it did occur and was due to patent prepared baby foods. In a subsequent conversation with the house physician, in answer to the question—to what is scorbutus among children commonly attributed in this hospital—he promptly gave the name of a proprietary food which is advertised upon and within every omnibus in London, at every railway station in all England, and whose name glares at one from the back cover of every sixpenny novel. This point will be of interest to our worthy and honored

president. Well do we remember his able plea before the Academy of Medicine in New York, that the tender life of the children of this country might be emancipated from the thrall of enterprising pap-peddlers. It is a significant fact that the country which furnishes most of the literature of scorbutus in children is the same which is posted from end to end with advertisements of proprietary foods.

As to the frequency of scurvy in America, Dr. Roger S. Tracy, Register of Vital Statistics of New York Board of Health, has very kindly copied off for the writer all deaths reported under scurvy from 1870 to 1890. In this twenty years, there have been 100 deaths from scurvy, reported to the office, of which sixteen have been in patients of five years or under. Of the sixteen fatal cases in children, twelve have been in infants (under two years of age). So it comes about, that of the class of cases which we are here and now considering, sixteen have been reported among the deaths of the last two decades in the City of New York. Some have doubtless gone astray in the records, because scurvy has been little considered and less thought of among the derangements of childhood and infancy; many of them, no doubt, have found their way to the list of purpura and acute rickets, some to hæmorrhagic exanthemata.

Surgeon J. S. Billings, librarian of the Surgeon-General's office, has kindly furnished seven references to cases of scurvy in children and infants. The first five cases are from England, the sixth from France and the seventh from America. This case on examination is not considered scurvy by the writer.

It would seem that England, and that means London in this case, is the source of most of the cases, and of the literature, of scurvy in children. To W. B. Cheadle and Thomas Barlow of Great Ormond street Hospital, is due the credit (the former) of "having first shown on clinical grounds the true affinities of this form of infantile cachexia," and (the latter) the anatomical nature of the diseases determined by post-mortem examination. Scurvy

is not in the index of Meigs' & Pepper's Diseases of Children (Edition, 1883); nor does it appear in the last edition of J. Lewis Smith's book.

The writer has held correspondence with twenty-five persons, members of this Pediatric section, and others, in New York and different large cities; the purpose has been to unearth any cases of infantile scurvy unreported and to learn the opinion of the gentlemen as to its frequency of occurrence. The answers have been prompt, to the point, of great service to the writer, and he here desires to express his obligations. All of these correspondents have vast opportunities for seeing cases in dispensaries, hospitals and asylums, as well as private practice. The majority of the answers contain such words (and with relief and satisfaction we record it) as "rare in children." "I know of very few cases of scorbutus"; "have thus far not seen any case of scurvy in children." "Have never recognized a case . . . nor do I know of any recognized case." "Have never seen a case in children in this city nor at the Juvenile Asylum where I pass in review 1,000 annually, and many are from the 'slums.'" "I think the cases extremely rare." "Cases of scurvy in children I have not seen." "During a service of two years, in which time 1,600 children have been seen, I have not met a single case of scurvy."

The purpose of this paper has been so modest that it may fairly be assumed that that purpose has been attained. Cases have been collected—eleven of them—and with pains to make the number represent the approximate frequency of occurrence. We must now await the report of the investigator for the intimate essential cause of the various lesions of scurvy. It is not a question of opinion; it is a question of facts. The whole line of sturdy soldiery must await the arrival of the aid who brings a message from him who holds his council amid culture ovens and flasks, dry plates and oil immersions.

So many apparent causative elements enter into the hæmorrhagic diseases and blood-dyscrasia, from drugs to bad food, that it seems idle to recount them; they rest

just where they did decades ago. The germ investigations have not rendered a final answer, though they seem soon to explain some of the group.

Finally, to dwell on such a topic as the present, to bring together rather more cases of scurvy than one expects are to be found, to, in a certain way, apply for the admission of a new disease into American literature, is somewhat similar to the player's role of villain; he may set forth upon the boards a most excellent villain, but after all, it is a gruesome business.

DISCUSSION.

DR. HOLT.—I have recently seen one case quite typical in its symptoms in which the diagnosis was confirmed by an autopsy. The history of Dr. Northrup's fatal case is almost the same as that of my case, and the lesions precisely those described by him. (See Case IX.)

DR. ROTCH.—That is to say, one of the starchy foods.

DR. NORTHRUP.—I should say not a food at all, but a *preparation.*

DR. ROTCH.—I would suggest here, in view of possible law-suits, the avoidance of the term "patent food." The manufacturers insist that it is not patent, but it is proprietary.

DR. NORTHRUP.—I should not call it a food at all, but a proprietary *mixture.*

DR. FRUITNIGHT.—In the last few weeks I have run across a case of this kind. She was a girl of five years belonging to the middle classes, but who having abundant food from some freak would not and did not eat any vegetables. The child had been sick about three weeks when I first saw her. The gums were spongy, tender and bleeding very readily. There were fugitive pains throughout the body, an herpetic eruption and some œdema of the left leg were present, with tenderness near the condyles of the flexors. The patient was put upon iron citrate, acid fruits and vegetable food and shortly recovered. In a large experience of many years this is the only case which I have seen. Furthermore, at the St. John's Guild Seaside Hospital, we have never seen a case of scorbutus, although, the number of cases treated is exceedingly large, and they are drawn from the poorest classes in the city, among whom, nutritious and milk food is lacking.

DR. JACOBI.—In Dr. Northrup's paper, replete with so much information, I missed one important fact, that is, the definition of what he means by scurvy. Is scurvy a complex of symptoms, the principal one being hæmorrhage, including hæmorrhage of the gums? Is the bleeding from the gums the characteristic feature of scurvy? This has been claimed, but, perhaps, incorrectly. Among the few cases related last year in Berlin, by Rehn, six had this symptom, one did not. Still it was claimed to be and was a case of scurvy. I have seen a case in a baby of five months in which there was no bleeding from the gums. The presence of the teeth may have something to do with the bleeding. My experience has been: no teeth, no bleeding from the gums. Ulcerous stomatitis, that form which I believe was first described in 1832 and which consists in the breaking down of the part of the gums adjoining the teeth, sometimes including the canine, is seen only in babies with teeth. Perhaps this disease has something to do with the anatomical condition around certain classes of teeth. In regard to the diagnosis and definition, it appears that no one has said up to this time what scurvy is. Many good and valuable writers are not able to say whether scurvy differs in any way from other hæmorrhages. I refer for instance, to that elaborate volume of W. Koch's, in *Deutsche Chirurgie;* it is entitled "Scurvy," and I find a few hundred pages on scurvy and a few short chapters where you have disquisitions on rheumatic purpura, hæmophilia, Werlhoff's disease, etc. He takes the ground that all belong to the same class. To say the least, the transition from one of these cases to another is too slight to make a differential diagnosis. The fact that in New York City so many cases have been reported as purpura and so forth, etc., shows that probably many of these cases have not been properly diagnosticated. Two cases I have seen this year—one was sent to me a few days before I sailed—it was a little baby with a few teeth, suffused gums and a few hæmorrhagic spots. The diagnosis of scurvy had been made. There were no hæmorrhages from the kidneys. There were spots all over the body. The child was treated by acids and improved rapidly. The other, a child of three years, was bleeding from the gums and from the intestines. I looked for the cause and found it. The child was very anæmic and diabetic, and when

the bleeding once stopped the child grew worse and died soon after. A number of cases are recorded, in which there was a tendency to have a large number of hæmorrhages from different organs and tissues. In most, there is a predilection to general hæmorrhage, as in typhoid fever, scarlet fever and rheumatismus nodosus. Now and then the kidneys, gums and other organs may bleed. In these, therefore, if we agree that we have no scurvy but only cases of hæmorrhagic diathesis in which the gums bleed, we know what we are talking about, but if not, then we are at sea. The cases described by Dr. Northrup here are similar. So far the microscopical investigations have been without a result. Large numbers of leucocytes are found in the blood-vessels, but to this time this condition has not been associated with any special bacillus. The doctor laid particular stress on the fact that this case occurred in a wealthy family. Children may be as much neglected and ignorantly fed in rich as in poor families. There is one peculiar fact in all cases, that as far as I know there were no hæmorrhages into the joints. The hæmorrhages sometimes extend over the epiphysis, but, as a rule, the joints are always intact. I would like the doctor to tell us whether he insists on calling scurvy only those cases in which the gums bleed or not, and if so, he must exclude the babies under six or seven months.

DR. NORTHRUP.—Dr. Jacobi is trying to lead me into deep water. I have carefully remained within the life line of facts, and have intentionally avoided discussion of the nature of the disease of which nothing is known. The cases here described correspond exactly with those described by Cheadle, Barlow and Eustace Smith.

As to the diagnosis, I should say the points that have thrust themselves before us most prominently in the recital of cases are the reliable ones, viz.: (1.) Pain on handling, located sooner or later in the thighs, also swelling, "rheumatism of the knees," as the mothers call it. (2.) Spongy gums. (3.) Rapid and marked improvement and recovery on administration of anti-scorbutics, oranges, etc.

DR. JACOBI.—I have no fault to find with the modesty of the author, but if no discussion was had, no benefit would accrue. Dr. Northrup says that he has many cases that agree perfectly with the articles in *Dr.*

Keating's Cyclopædia. We are not here for the premeditated purpose of agreeing; but to get along a little further in the line of research. I did not find Dr. Northrup in deep water before, but I do now. He claims the principal symptom of scorbutus is the subperiosteal hæmorrhage. This cannot be, for we find it is the first symptom in syphilis, often. Secondly, we find subperiosteal hæmorrhage in the new-born over the cranium. From what cause? From slight pressure. We find subperiosteal hæmorrhage even on the occipital bone where there is but very little pressure, because the blood-vessels are very thin in the new-born and are but insufficiently protected by their shallow grooves. It is also thus over the long bones, so the subperiosteal hæmorrhage cannot be considered a characteristic symptom of scurvy. I believe you must look for other causes, whether it is an invasion into the blood-vessels, whether it depends upon anatomical causes, or is a result of defective enervation, perhaps, always resulting from improper food. The blood in any condition will not extravasate unless the blood-vessels be changed, either by disease or congenitally. In the latter case, we may have a disproportion between the heart and the blood-vesels or unduly small arteries compared with the other blood-vessels. Thus, hæmorrhage may occur. So I do not think we are correct in diagnosticating scurvy from that symptom alone. Dr. Northrup spoke of the femora. It is more the femur than the tibia that is affected. Is it not so?

DR. NORTHRUP.—Yes.

DR. ROTCH.—Dr. Northrup does not mean hæmorrhage of the gums and thighs is enough, but these are two of the diagnostic symptoms of scurvy.

DR. JACOBI.—Are we entitled to call all forms in which hæmorrhages show themselves, morbus maculosus, rheumatic purpura, etc., scurvy? May these be classed in the same category with scurvy?

DR. NORTHRUP.—I have seen that article of Dr. Koch's, referred to. I stop where my facts stop. I want to say that hæmorrhages from the gums and subperiosteal hæmorrhages mean scurvy; I have collected twenty-five cases and reported them. In this case (Case I,) I felt in an instant that it was scurvy, and the condition of this child I call an indication to the general practitioner to be on the look out.

DR. JACOBI.—No one has collected so many cases as Dr. Northrup. While I do not feel like admitting that scurvy is different from the other forms I thought the proof might come out in this discussion, where there is plenty of material, and those who use the microscope might show what the Europeans have been unable to determine. And I hope that some of the members here present may be able to prepare a scheme for an exact diagnosis. I hope those gentlemen who are experts, informed and able, will study the conditions of the blood-vessels in these cases. Is there a foreign substance present in these cases? Is there a bacterium always present? Do the conditions exist in similar diseases?

DR. SMITH.—Having been connected for many years with asylums, I think this is not so rare a disease as supposed, but that we have been treating cases from time to time perhaps under a different name. We are now seeing cases of rötheln which we used to call epidemic roseola. I think it might be well to make investigations during the coming year, and discuss this disease at our next meeting.

A CASE OF CONGENITAL MALFORMATION OF THE HEART.—ATRESIA OF THE PULMONARY ARTERY WITH PERSISTENCE OF THE FŒTAL CIRCULATION.

BY WILLIAM TRAVIS HOWARD, JR., M.D.,

Baltimore.

*I am indebted to the courtesy and kindness of my friend Dr. L. Ernest Neale, in whose practice it occurred, for the autopsy and the history of the following case:

The father is a merchant; both parents are healthy. The mother is a multipara, and the delivery was normal. The patient was small and weak, weighing only five and three-quarters pounds at birth, and lived three months

*The histological portion of the work was done in the Pathological Laboratory of the Johns Hopkins University and Hospital.

and six days. It was quite cyanotic, and was a typical "blue baby."

On the second day, it was found that the rectum was imperfect. On the third day Dr. L. McLane Tiffany operated successfully for the relief of this, no anæsthetic being used. During the operation, the child was held by the heels, its lower extremities, buttocks and abdomen being nearly black, and its chest, head and upper extremities quite blue; its pulse and respirations very rapid.

The operation was followed by marked shock and collapse, with cyanosis, lasting two days. Under stimulation and careful nursing it improved and would be of quite a natural color for a day at a time. It, however, frequently had attacks of cyanosis with collapse. Its pulse was always rapid. Congenital heart disease was naturally suspected, but after careful and repeated examinations no murmur could be detected; an exact diagnosis as to its seat was not ventured upon. The heart-sounds were loud and clear.

The child gradually wasted and became weak, slight convulsions and cervical opisthotonos were observed. Towards the last, the hydrocephalic cry was heard. In spite of careful attention and nursing, the child gradually sank.

The following notes are taken from my autopsy records:

Autopsy.—Body wasted, small for age, 51 cm. long. There is no marked cyanosis. The cheeks are sunken, the fingers and toes somewhat clubbed. There is no dropsy. The anus is represented by a slit 5 cm. long. The head measures 54 cm. in circumference. On the removal of the skull-cap about 10 cc. of creamy pus escapes from the right side. There is basic and cortical meningitis, and encephalitis, with ependymitis, and distension of the ventricles with pus.

Heart.—The peri- and epi-cardia are pale, but otherwise normal; there is no effusion. The heart is asymmetrical in shape, the right ventricle being quite small and very strongly contracted.

The external appearance of the right ventricle is smooth, of an irregular shape, measuring from its junction with the right auricle to its apex 3 cm. and transversely at the base, 3.5 cm. Its wall is strongly contracted, but not hypertrophied, and measures in thickness near the valves 3 mm., and in the middle portion, where it is thickest, 11 mm. The musculature of the wall is of a light red color and of firm consistency. The inner surface of this ventricle measures from apex to base 12 mm. and its greatest transverse diameter is 17 mm. Its surface is smooth, and its cavity, which is rounder than normal, is nearly filled by an irregularly round fibrous mass, grayish-red in color, with white opaque edges, measuring in its greatest diameter, the transverse, 15 mm. It is nowhere adherent to the ventricular wall or to the valves, and is freely movable over the cordæ tendinæ, which runs through it.

The right auriculo-ventricular orifice is small, measuring only 13 mm. in circumference. It is closed by only two valves, which are somewhat thick, with their free borders elevated, but present no vegetations.

The inter-ventricular septum is perfect, but appears to run somewhat obliquely from left to right, making the right ventricle smaller than the left. The pars membranasa is perfect.

There is complete atresia of the orifice of the pulmonary artery at its origin from the right ventricle, the conus arteriosus being represented by a small depression of the size of a pin's head.

The point of atresia is situated at the position of the semi-lunar valves, the only traces of which are three small folds running several millimetres in the length of the artery. The atresia is complete, and the pulmonary artery above this point is narrowed and atrophied.

Its walls are smooth and it branches 15 mm. above the ventricle, and 2 mm. below the opening of the ductus arteriosus, which is 1.5 mm. in diameter.

The diameter of the pulmonary artery just above the point of atresia is 3 mm., and of the left pulmonary artery at the opening of the ductus arteriosus, 1 cm.

Right auricle is considerably dilated and hypertrophied. Its greatest diameter is the transverse, which measures 5. cm. Its walls average from 1 to 3 mm. in thickness, its inner surface is smooth, and the musculi pectinati are well developed. The orifices of both venæ cavæ are a little dilated, but are otherwise normal. The auricular appendage is large, measuring 3.5 cm. in length, the last 2.5 cm., being of fibrous tissue. The fossa ovalis is deep, and the foramen ovale is large, measuring 12 mm. in diameter. It is closed imperfectly by the Eustachian valve, which is represented by a thin membrane, and is complete, except at its inferior border, corresponding to the fossa ovalis, thus forming a more or less triangular opening between the two auricles.

This orifice when stretched forms a round opening 3 mm. in diameter.

Left ventricle is relatively large, globular in shape, its surface smooth, the musculature of its walls of a light red color and firm consistency. The length of the ventricle from the aortic ring to the apex is 2.5 cm., the thickness of its wall 9 mm.

The endocardium is smooth, and the musculi papillares well developed.

The segments of the mitral valve are normal and the circumference of the mitral orifice 4 cm. The middle leaflet of the aortic valve is thickened, and its free border retracted, the posterior leaflet less so. The circumference of the aortic orifice is 22 mm.

The left auricle is slightly dilated; its wall measures 1 mm. in thickness. The septum between it and the right auricle is imperfectly closed by a valve-like membrane, forming an opening which, when stretched, measures 3 mm. in diameter.

The auricular appendage is normal. The pulmonary veins enter by a double opening.

The Aorta.—The ascending arch of the aorta is larger than normal, measuring 2.5 cm. in circumference. It, with the innominate artery, appears dilated; both are free from patches.

The ductus arteriosus is given off 2 cm. above the aortic valves. Its margins are puckered, and its lower margin presents a triangular elevated ridge.

The orifice is more or less triangular in shape and measures 3 mm. in its greatest diameter.

The ductus is not funnel-shaped, though its aortic is larger than its pulmonary opening.

The weight of the heart is 31 grmms.

Lungs.—There is no effusion in the pleural cavities. The lungs are emphysematous, and their margins appear thickened. Muco-pus is squeezed from all the bronchi. There are small scattered areas of atelectasis.

The abdomen presents nothing abnormal.

The liver, spleen and kidneys show nothing of special interest.

The rectum opens into anus by a constriction 4 cm. and just below it 2.5 cm. There is absence of the coccyx; the sphincter is well preserved, and the operation wound entirely healed.

Microscopical examination of fresh frozen sections shows fatty degeneration of muscle fibres of the left ventricle, fatty infiltration of the liver cells, fatty degeneration of the epithelia of the kidneys, with thickening of the walls of the glomerular capillaries, and round and epithelial cell in the capsular spaces.

Hardened sections through the roots of the lungs show a slight dilatation of the bronchial arteries. The pulmonary arteries are of about normal size. There is a general bronchitis, and a slight diffuse interstitial pneumonia, best marked at the margins of the lungs. A few small areas of atelectasis are seen.

Anatomical diagnosis. Complete atresia of the pulmonary artery at its origin, strong contraction of the right ventricle, presence of but two segments at the right auriculo-ventricular orifice; patency of the foramen

ovale and the ductus arteriosus, thickening and retraction of the middle and posterior segments of the aortic valve, hypertrophy of the *right* auricle and the *left* ventricle. Bronchitis with interstitial pneumonia and emphysema of both lungs, and slight dilatation of the bronchial arteries. Fatty degeneration of the heart and kidneys, and fatty infiltration of the liver. Acute purulent ependymitis and encephalitis, with basic and cortical meningitis.

There are many interesting points in this case, but only those relating to the heart will be considered here.

The most striking features are the complete atresia of the pulmonary artery with patency, but not dilatation, of the ductus arteriosus, and patency of the foramen ovale, without imperfection of the ventricular septum.

Undoubtedly there was in this case persistence of the fœtal circulation. Ordinarily one would find great dilatation of the ductus arteriosus and the bronchial arteries. But the child did not grow, and before death weighed a quarter of a pound less than at birth, hence, there was no need for the dilatation. The whole process is a congenital malformation, and as such is especially interesting in being associated with congenital imperforate rectum.

Had the atresia of the pulmonary artery been gradual, as from an inflammatory process, there must have necessarily been an hypertrophy of the *right* ventricle *pari passu* with the increasing obstruction to be overcome. Here the right ventricle is not hypertrophied, but is strongly contracted. So we would class this case with the great majority of such cases as due to a developmental error. In attempting to explain how these errors may occur, it is necessary to consider the development of the normal heart.

The systemic and pulmonary aortæ are formed by the junction of two longitudinal projections springing from the opposite flattened sides of truncus arteriosus.

These projections arise from slits in the lumen on each side of the truncus, grow and gradually coalesce, dividing

the truncus into two triangular portions. The separation is marked on the external surface of the truncus by two furrows, which are analogous to the two inter-ventricular sulci. The entire process in the truncus goes on entirely independently of the development of the inter-ventricular septum, but at the same time. Finally, the aortic septum, or septum of the truncus enters the ventricular cavity and uniting with the inter-ventricular septum furnishes the portion known as the pars membranasia, thus completing the separation of the heart cavities, the aorta belonging to the left and the pulmonary aorta to the right ventricle. In the fully developed heart the pars membranasia represents the place where the closure took place last. In reptiles, this portion is not closed off, and an opening between the ventricles is left, the foramen Panizzæ.

This fact is interesting as showing the ease with which in so many cases of congenital narrowing and atresia of the pulmonary artery, compensation is accomplished by the failure of closure of the foramen Panizzæ by the septum of the truncus.

On the other hand, many cases in which both the atresia or narrowing, and the failure to close this foramen occur, may be explained by assuming that the same error in development caused both lesions.

The semi-lunar valves are first seen as four projecting masses of gelatinous substance covered with endothelial cells, one from each aspect of the narrowed portion of the truncus, at the fretum Halleri. When the division of the truncus into the aorta and the pulmonary artery takes place two of these projections are halved, so that each vessel receives a whole projection and two halves.

Normally, each whole projection develops into the middle segment or leaflet, and the two half projections become the anterior and posterior segments of each semi-lunar valve. This holds good for both the aorta and the pulmonary artery. So there are several possible modes for errors in development in this situation. (1) There may not be the proper number of projections

formed. (2) One or more of these projections may be imperfectly formed. (3) In consequence of either of these errors, there is not the normal amount of valve tissue left on one or both sides, after the division of the truncus to form the normal valve segments. (4) Perfectly formed and divided projections might unite and grow together, and, steadily contracting, cause narrowing or atresia of either or both orifices, with or without imperfect ventricular septum accordingly as the septum of the truncus arteriosus does or does not form the pars membranasia.

In my case the middle or undivided segment of the aortic valve was thickened and retracted, and the posterior segment somewhat less so. This with the complete atresia of the pulmonary orifice would suggest an imperfect development of the primitive valvular projections as well as faulty division. Another element that may come in here is the chance of an oblique division of the truncus arteriosus at the expense of the right side.

This error would make the pulmonary artery at its origin small, and would cause faulty division of the valvular projections and consequent atresia of the pulmonary artery and widening of the aorta in its arch. This would explain the present case. In cases of narrowing and of atresia of the pulmonary artery with the left ventricle much larger than the right ventricle, there is probably an oblique division of the primitive ventricle by the inter-ventricular septum, in addition to the above-mentioned errors.

In most cases of pulmonary artery atresia the ductus arteriosus is very much dilated and often funnel-shaped.

Here it is not larger than normal, but still patent, though it should normally close by the fourteenth day. As the foramen ovale is also patent, the fœtal circulation has persisted. In this connection it is interesting to call attention to the point that the right ventricle was strongly contracted, but not hypertrophied, while the left ventricle was hypertrophied and somewhat dilated.

The absence of a cardiac murmur during life is explained by the facts that the fœtal circulation persisted and that there is no murmur produced by the heart of a normal fœtus.

In the light of these observations, when symptoms of cardiac disease and obstruction to the circulation are met with in the newly born without a cardiac murmur, one must strongly suspect the existence of the cardiac conditions described in this case.

DISCUSSION.

DR. HOLT.—This case seems to me an unusual one in that there was no inter-ventricular opening, not even a patent foramen ovale.

DR. SMITH.—Several years ago, I collected all the cases of cyanosis which I could find, the number amounting to 162. I found in an analysis of these cases the most common lesion was an absent rudimentary, impervious, or partially obstructed pulmonary artery (97 cases), so that the only way in which the blood could reach the lungs was by a circuitous route from one side of the heart to the other by the foramen ovale or through the inter-ventricular opening. Had this child lived, it would have been cyanotic, especially in times of excitement. Sometimes cyanosis does not appear for a month or more till some excitement occurs and then it is continuous.

AN IMPROVED METHOD OF PERFORMING ARTIFICIAL FORCIBLE RESPIRATION.*

(With Exhibition of Instruments.)

BY J. O'DWYER, M. D.,

New York.

The subject to which I will briefly call your attention is a modification of the method of producing artificial respiration as devised by Dr. Geo. E. Fell, of Buffalo.

In the performance of artificial respiration by any means, it is important to remember that all we have to do is to get air into the lungs and to give it sufficient time and room to escape, the power generated and stored up in overcoming the resistance to inspiration being amply sufficient to carry on expiration.

The Sylvester and similar methods which aim to imitate natural inspiration by expanding the chest are extremely inefficient, simply because the chest cannot be expanded to any considerable degree, without contraction of the inspiratory muscles and especially of the diaphragm. Dr. Fell's method, which is identical with that practised in the laboratories on lower animals, consists in forcing air into the lungs by means of a foot bellows—the connection between the bellows and the lungs being effected by means of rubber-tubing attached to a canula in the trachea or to a mask applied over the mouth and nose. To both of these means of making the connection, there are serious objections.

In one, not only has tracheotomy to be performed but the wound around the canula must be made air tight, and the trachea either tied or tamponed above the incision, to prevent the air from escaping upward through the larynx.

In forcing air through the mouth or nose, of an insensible patient, the tongue, unless secured, is almost certain to cause obstruction, or the vocal cords may be forced together by the in-rushing air, and act as a valve as in paralysis of the abductor muscles because there is no expansion of the glottis as in normal inspiration. Should the larynx become obstructed from any cause, the stomach will be inflated instead of the lungs.

I have devised a set of tubes, which I think will overcome these difficulties by establishing direct communication between the bellows and the lungs through the natural passages.

The laryngeal portion of the tube, as you will see, is conical in shape and is intended to tampon the larynx below the vocal cords, so that no air can return beside it.

This portion of the larynx is almost cylindrical in shape, considerably smaller than the trachea and therefore more easily plugged than either the triangular shaped rima or the much larger cavity above.

The set consists of two long tubes, one for children the other for adults, and five laryngeal tips, the lower portion of the latter being surrounded by grooves to allow the vocal cords to aid in holding them down.

The proximal portion of the long tube has two openings —one for inspiration to which the rubber-tubing connecting with the bellows is attached, the other to be controled by the thumb of the right hand.

Those familiar with intubation in croup will find no difficulty in the introduction of these tubes in children; but in adults it is not so easy to use the finger as a guide, owing to the greater distance of the larynx from the mouth. On the cadaver, I have found it much easier to properly place the tube without any guide, by carrying it well down towards the œsophagus, and then coming forward in the middle line with downward pressure.

The principal danger attending this operation is injury to the lungs from forcing air in and not allowing sufficient time for it to escape, resulting in over-distension and rupture of the pulmonary vesicles.

The best safeguard against this accident is making the respirations slowly—ten or twelve to the minute—and watching the movements of the patient's chest, which should be uncovered, with the exception of the closely fitting under-garment.

The value of this method of performing artificial respiration in man has been amply demonstrated by Dr. Fell in a number of cases of opium poisoning successfully treated.

Dr. Horatio C. Wood, in his paper on Anæsthesia, read before the International Medical Congress, speaks as follows of its value in resuscitating animals apparently dead from ether and chloroform.*

*1st vol. of the transactions.

"The most remarkable results which I have reached in bringing about recovery of animals to all ordinary intents and purposes dead, were obtained through the use of artificial respiration. Thus, I have seen an animal in which no respiratory movements, whatever, had taken place for two minutes, and in which no movements of blood had occurred in the carotid, and in which the heart had therefore ceased to beat rapidly and permanently, restored by artificial respiration. At one time in these researches, it appeared as though after any dose of chloroform by inhalation, the animal could be resuscitated by artificial respiration, even though heart and lungs were completely paralyzed by the drug; but finally, I did find a case in which, after the animal had repeatedly been apparently killed and resuscitated, artificial respiration failed."

After discussing the value of drugs in poisoning by ether and chloroform, Dr. Wood continues as follows:

"The one measure which in practical value far surpassed all others for the restoration of the dying animal was artificial respiration and I have no doubt that a great majority of the deaths which have occurred in man from anæsthesia, might have been avoided by the use of an active artificial respiration. The difficulty with artificial respiration, as it has been hitherto practiced on man after the Sylvester or other method, is its inefficiency, whereas, the artificial respiration which I used was very active indeed; much more efficient than natural breathing in causing circulation of air in the lungs, and, therefore, in removing excess of the poison from the residual air in the lungs and from the blood."

Artificial respiration is called for, not only in all forms of narcotic poisoning, but may also prove useful in those cases in which the inspiratory muscles are rendered temporarily useless, as if paralyzed by spasmodic contraction such as strychnine poisoning, puerperal and other forms of eclampsia, and also in acute pulmonary obstruction from various causes.

In the latter class of cases, I made some experiments six or seven years ago at the New York Foundling Asylum,

using the laryngeal tube I here show you, through which air was forced into the lungs by means of a rubber bag.

During the past winter, I tried the same instrument in one case with the bellows attached, and demonstated one thing at least, viz.: that we can breathe for a patient, while still perfectly conscious, and keep it up for several hours after he has lost the power to breathe for himself.

Another use to which these tubes may be applied is to prevent blood from entering the lower air-passages during operations in or about the mouth, while, at the same time, affording a free passage for the air to and from the lungs.

For this purpose, a lateral curve of the proximal portion would be necessary to prevent interference with the operation.

DISCUSSION.

DR. NORTHRUP:—We must look out for Dr. O'Dwyer ; he is always giving us something better than he promises. One Doctor can easily work this apparatus on a child for six or seven hours. I can verify the statement that "A single forced inflation cannot break the lungs, no matter how violent it may be."

DR. BOOKER:—Would this apparatus be of any use in absence of respiration in the new born?

DR. O'DWYER:—It might be in case it was considered desirable to keep up artificial respiration for a considerable length of time but I have found the mouth to mouth method of inflation amply sufficient in the asphyxia of the new-born.

ON THE ASSOCIATION OF CONGENITAL WRY-NECK, WITH MARKED FACIAL ASYMMETRY. *

BY WILLIAM OSLER, M.D.,

Johns Hopkins Hospital, Baltimore, Md.

WILKS * speaks as follows of this condition: "In reference to wry-neck there is a fact worthy of observation and further investigation; that in young persons subject to this affection the head and face on the contracted side do not develop as on the other, and in consequence there is a want of symmetry in the countenance when narrowly examined from the front. One eye is slightly lower than the other, and the whole of that side of the face and head is smaller than on the other. In a lad lately in the hospital with heart disease, a wry-neck existed from infancy, and this remarkable want of symmetry was very evident. In a young lady patient, also, who is otherwise well grown, this disproportion of the two sides of the head and face is clearly shown. It may be asked whether this is due to some failure of nervous power having its foundation in the same cause which produced the wry-neck, or whether the contracted muscle itself exerts an influence on growth, and, if so, whether the division of the sterno-mastoid would allow development again to proceed." The references to it in the literature are scanty, and it has even escaped the notice of that most observant of observers, Gowers.

* "Diseases of the Nervous System," 1st edition, 1878, p. 454.

Bradford * mentions a child of six months, which was born with a lack of symmetry of the cranium, affecting the frontal bone on the right side. The face and head in every other respect were symmetrical. The head was habitually carried in the position of wry-neck with the chin turned to the right, exactly in the position to suggest the explanation by the mother that the left side was heavier than the right, and the head was held crooked.

Krummacher † reports two cases; in one, a child aged ten with right-sided torticollis, the face showed marked asymmetry. In the second the child, aged twelve, at about the age of two years fell and had paralysis of the extremities on the right side. The sternal portion of the right sterno-mastoid was contracted. The facial asymmetry was striking; very little difference in the extremities; no special asymmetry in the two sides of the skull.

Stanley Boyd ‡ reports a case of a girl, aged nine years, in whom the torticollis was noticed shortly after birth. The right half of the face was distinctly smaller than the left. Careful measurements and the accompanying photograph showed the great difference which existed between the two sides, not only of the face but of the skull.

By far the most important communication on the subject is by C. H. Golding-Bird, § who reports six cases of congenital wry-neck with facial hemi-atrophy.

The following case has come under my observation :

Margaret G., aged fifteen, a well-grown, healthy looking girl, with good family history. She was well as a child. Nothing wrong was noticed until her tenth year. On the 18th of December, 1886, she fell and cut her chin, and the mother states that for two weeks she held her head turned to the right, and for a year or more after this she had wry-neck. The following year Dr. Tiffany operated, and there was temporary benefit. Subsequently by gymnastic exercises she improved very much. The mother is positive that the child had not wry-neck when young. The asymmetry of the face has been noted for several years, and is thought by the mother to be progressive. A photograph taken at four and a half, full

* *Boston Medical and Surgical Journal*, vol. cvi., 1882.

† "Berlin Thesis," 1889. *Zur Aetiologie d. Schadel-Asymmetrie beim Angeb. Scheifhalse.*

‡ *Illustrated Medical News*, London, 1889.

§ *Guy's Hospital Reports*, vol. xlvii, 1890.

face, shows complete equality of the sides; no lowering of the eye or eyebrows. A second picture at eight and a half shows both sides fairly equal and the eyes on the same level.

Neck; circumference, 30.5 cm. The left side is distinctly fuller than the right; scarcely perceptible scar above the sternal attachment of the sterno-mastoid. The outline of the sterno-mastoid on the right side is plainer than on the left when the head is straight and the depression between the two portions is better marked. The muscle on the left side is distinctly larger and fuller, and is of greater breadth; particularly well seen when the head is rotated to the right. The clavicular part is full and strong, and there is no trace of the division between the sternal and clavicular portions. When the head is rotated to the left the sternal portion stands out prominently and well. No difference apparent in the trapezius of either side.

Movements of the head seem free, but she rotates to the left rather more than to the right. The face shows marked asymmetry. The measurements are as follows: On the left side, from below the lobe of the ear to the angle of the mouth, is exactly 10 cm.; corresponding measurement on the other side is exactly 9.2 cm. From the outer canthus to the tip of the antitragus: right, 8.4; left, 8.8. A horizontal line through the middle of the left pupil passes at the margin of the upper lid of the other eye. A line passing at the margin of the lower lid of the left eye passes through the middle of the pupil of the right. The right eyebrow is on a distinctly lower level than the left. The nose is straight.

The whole cheek, including the malar bone and zygoma of the right side, is distinctly smaller than the left; no difference in the ears. Pupils are equal, react alike. Laughs on both sides of her face equally. Both frontals move well. A little flattening perhaps in the right temple.

The measurement of the head, 36.5 cm. Nothing of note in the hands and arms. The teeth are well formed, equal. No molars in the lower jaw on the right side. The palate is well formed; the sides of the tongue equal.

The question arises whether this was really a case of congenital wry-neck. The mother is quite positive in her statement that the child held her head perfectly straight until after the accident at her twelfth year. Golding-Bird mentions that in all but one of his cases he was

informed that the deformity had only been recently noticed, but an appeal to the photograph album in several instances showed that the deformity had existed long before the parents had noticed it. In our patient the photograph taken at four and a half and the photograph at eight and a half years show no asymmetry, certainly not in the earlier one. In the picture at eight and a half some have said the sides looked equal, others that the left side looked the smaller.

Facial asymmetry quite evident to the observer may be overlooked by the parents and friends, who have been familiar with the appearance of the child from infancy. Under these circumstances it is only necessary to show them the reflection of the face in the looking-glass, which brings out the asymmetry between the sides in a striking manner.

All of the cases on record have been on the right side.

Slight grades of facial asymmetry are exceedingly common, probably quite as common as asymmetry of the skull, but it is only when extreme and progressive that the term hemi-atrophy can be applied to it. The condition evidently is quite different from Romberg's facial hemi-atrophy. In none of the cases reported has the unilateral wasting been progressive, nor has it proceeded to the same grade as seen in this affection. The skin is not observably changed, and there does not appear to be the same loss of subcutaneous tissue, nor is there wasting of the sebaceous follicles, nor any change in the nutrition of the hairs. The differences in the bones and of the muscles may, however, be very striking in the form at present under consideration.

In the cases with torticollis the condition is rather, as Wilks suggests, as if the face on the affected side had not developed proportionately, so that it is arrest of growth rather than an actual atrophy.

A perfectly satisfactory explanation of this asymmetry and of its relation to torticollis has not yet been offered. Eulenberg (quoted by Golding-Bird) has suggested that the position of the neck interfered with the vessels and nerves passing to the head and so arrests the nutrition, but this does not seem very likely. Golding-Bird advances the view that the facial hemi-atrophy is not a consequence of the torticollis, but an integral part of the affection, both having a common central origin, which he believes is a primary polio-encephalitis. He states that "bearing in mind the exact similarity between congeni-

tal torticollis and a case of infantile paralysis with talipes equinus, the conclusion is, to my mind, all but inevitable that "caput obstipum," with its contractured sterno-mastoid, wasting of the facial muscles and soft parts, and even of the bones, forms an exact copy of talipes equinus with contractured calf-muscles, ill-nourished soft parts, and in many instances shortened bones, and must have a similar origin." It certainly appears more reasonable to think that the hemi-atrophy and the shortened muscles are both the expression of some central lesion, but it is difficult to understand, from our present knowledge of cortical localization, exactly where the mischief could be.

The facial condition is apparently not progressive. As already mentioned, it is a question whether it is a condition of atrophy of the face or whether it is not really an arrest or imperfection in the development. If the former it would, as in Romberg's type of hemi-atrophy, produce in time more serious alterations than have been present in any of the cases yet recorded. The history and the photographs in my patient suggest that the wry-neck and asymmetry may have developed after the injury. The facial condition is such as might be produced by arrest of development, but how brought about and in what way related to the torticollis is not very clear.

GAVAGE IN THE TREATMENT OF PERSISTENT VOMITING IN INFANTS.

BY CHARLES G. KERLEY, M.D.,

Resident Physician, New York Infant Asylum, Mt. Vernon, N. Y.

A NUMBER of cases of persistent vomiting occur every year in the New York Infant Asylum, largely in delicate and athreptic infants, either with or without intestinal complications—cases in which diet, ordinary therapeutics, stomach-washing and other measures prove fruitless. In the treatment of some of these obstinate cases of vomiting by stomach-washing, especially in those with high temperature and urgent thirst, it has long been our custom to in-

troduce an ounce or two of boiled water into the stomach, after the completion of the washing, and allow it to remain. It was observed that the water so left behind was rarely rejected, when whiskey and brandy, largely diluted, and light foods of various kinds were vomited when given by spoon or bottle within an hour or two after the washing.

It occurred to the writer that, if water given in this manner could be retained, partially digested food and diluted stimulants might be kept down, if given in the same way; therefore it was determined to see what could be done in the way of systematic forced feeding by means of the stomach tube in the treatment of these cases.

After ten or twelve had been treated in this manner, Dr. Holt informed me that Tarnier and other French writers had employed similar measures in the management of premature and weakly infants. As the method and object is practically the same, the term Gavage or forced feeding, which was applied by Tarnier to this procedure, is adopted, although it was never employed by him in the treatment of vomiting.

A search through the literature of the subject shows that Gavage has been used successfully, not only, as by Tarnier in premature and delicate infants, but after operations for hare-lip, tracheotomy, intubation, etc., but not, so far as I have been able to learn, by any writer to combat the symptom vomiting.

As a rule, the children upon whom our observations were made were delicate; a few had marked athrepsia; in no case was forced feeding employed until other measures had been found unavailing. In many instances the forced feeding was alternated with the bottle or spoon, in order to discover whether the success was due to the method, or whether the irritability had been controlled.

The apparatus employed was that ordinarily used for stomach-washing, with this exception: instead of the soft rubber catheter, Pain's No. 1 adult nasal feeding tube was used. The advantage of the feeding tube over the catheter lies in the fact that the former contains two eyes while the latter has but one. This shortens the duration

of the operation about one-half—a decided advantage in some cases. The feeding tube is attached by a small glass tube to a common rubber tube two and one-half feet long, with a funnel holding three or four ounces at the end.

During the operation, the child should be held in a half reclining posture, supported by the nurse's right arm, and not held erect as for stomach-washing; the tube should be introduced rapidly, the quantity to be given poured into the funnel, which is then raised as high as possible, and the tube compressed and instantly withdrawn as soon as the funnel has emptied itself into the stomach.

Usually diluted stimulants and predigested foods were given, in quantities ranging from ½ to 2½ ounces, every two or three hours. Twenty cases were treated in this manner; the forced feeding was employed in all one hundred and thirty-two times. A portion of the amount given was vomited eleven times, the entire amount twelve times; but after one hundred and nine feedings there was no vomiting.

The following cases are taken as illustrative examples: I am indebted to Drs. E. C. Wendt and L. Emmett Holt, Visiting Physicians, for use of cases, and to Dr. H. E. Tuley, Senior Assistant Resident Physician, for his valuable assistance in carrying on the work.

CASE I.—*Persistent vomiting, uncontrolled by diet or mechanical means, immediately and permanently relieved by forced feeding. Second attack two weeks later similar management; similar result.*

Fairly well-nourished male infant (colored), aged five months, nursed by mother until ten weeks old, when the nursing was stopped on account of persistent vomiting. The mother's milk was examined and found deficient in both fat and albuminoids; microscopic examination negative. Means for improving the milk attempted but unsuccessful. The nursing stopped entirely and put on the partially peptonized milk, which was retained fairly well; received two ounces every two hours, and vomited but once in two or three days.

The patient gained in weight and seemed fairly well until the middle of July, when the vomiting increased, averag-

ing two attacks in twenty-four hours, when the whole amount given was rejected. The stomach was washed frequently, and the amount given at a feeding gradually reduced to three or four drachms. The diet was also changed to malted milk—from one-half to one ounce given at a feeding. The careful diet, stomach-washing with doses of one grain of cerium oxalate in one-half drachm of chalk mixture, given hourly, were ineffectual. Was treated in this manner for five days, and on the sixth day of illness vomited everything. Greatly prostrated; pulse feeble; some cyanosis; temperature and stools normal.

As the malted milk had been vomited several times during the morning, when given by spoon or bottle, this was selected for the forced feeding; stomach not washed; and at three o'clock P.M., one and one-half ounces given by tube; two ounces given in this manner at 4.20 and one ounce at 7.30; all retained; no inclination to vomit. At 9.40 vomited the whole amount given, one and one-half ounces during the withdrawal of the tube; but retained the same quantity given in the same manner three minutes later. The temperature and stools normal. During the night fed one ounce by spoon every two hours, and vomited three times, everything taken, unchanged.

2d Day.—Temperature and stools normal. At 8 and 10 A.M. fed one ounce of malted milk by spoon, and vomited all as soon as taken. Stomach washed at 11 A.M. Some mucus and few curds removed. In forty minutes was given one ounce of malted milk by spoon; vomited all in ten minutes. The spoon-feeding and vomiting repeated at 1.20. One hour later was given one and one-half ounces by tube; no vomiting followed, and at 4.20, before feeding, the contents of the stomach were syphoned out. Some mucus and few curds removed. Was then given two ounces of malted milk by tube, which was retained. During the night took one and one-half ounce by spoon, every two hours, without vomiting.

During the next week from sixteen to twenty ounces of the malted milk was taken by bottle in twenty-four hours; no rise in temperature during the time; no vomiting; bright and playful.

At the commencement of the second week, however, a second vomiting attack came on, and for sixteen hours everything taken was vomited; malted milk, wine-whey, whiskey and water were tried. The stomach was washed and found empty, and in one hour one ounce of malted milk given by spoon; vomited at once; repeated the feeding in

two hours, and all vomited as soon as taken. One hour later, no medicine having been given, one and one-half ounce of the malted milk given by tube and retained; two ounces given in this manner two hours later and retained. During the remainder of night took and kept down one and one-half ounce by spoon every two hours. No more trouble followed, and at present report is gaining from five to twelve ounces weekly on the malted milk.

CASE II.—Delicate, bottle-fed male infant, aged six months; was taken sick the second week in July.

1st Day.—Temperature, 99° to 101°. Six large thin stools. Vomited twice the malted milk upon which he had been gaining in weight slowly. The diet was changed to barley-water and wine-whey; given one drachm of castor oil, ten grains of bismuth hourly, and one-fourth grain of Dover's powder every two hours.

2d Day.—Temperature, 100° to 102°. Fifteen green mucous stools, streaked with blood. Colon irrigated with four pints of the tannic acid solution at 110°; other treatment the same; the food taken well.

3d Day.—Temperature, 101° to 102°. Ten stools, same character; no essential change; treatment continued.

4th Day.—Temperature, 100° to 101°. Six stools, same character. Bismuth stopped, and instead given one-half grain of resorcine in one drachm of chalk mixture every two hours. The resorcine stopped after the fifth dose, on account of vomiting, and bismuth in ten-grain doses hourly resumed. No more vomiting, but the nourishment was taken very poorly. If more than two teaspoonfuls of the wine-whey or kumyss was given, retching and vomiting followed.

6th Day.—Temperature, 101°. Eleven stools, same character. Almost impossible to give any form of nourishment, but two drachms taken at a time during the early morning.

Stomach not washed, and at 10.30 A. M., ten drachms of the Mellin's food, without milk, was forced and retained. In two hours one and one-half ounce of the completely peptonized milk given in the same manner—repeated in two hours. No vomiting. At the next feeding, 4.30, one-half ounce of the milk was fed by the spoon; no more could be taken. During the remainder of the afternoon and night, one-half ounce was taken every two hours.

7th Day.—Temperature, 101° to 103.8°. Six stools, same character. At ten o'clock A. M. one ounce of the completely peptonized milk was forced and retained. Two hours before, but one-half ounce could be given by spoon.

The forced feeding repeated at 12 noon; one and one-half ounces introduced and kept down. Later, one-half ounce of food was given by spoon or bottle every two hours—one thing was taken as well as another—but if the fifth drachm was given the whole feeding was vomited at once.

Treatment continued; baths *p. r. n.*

8th Day.—Temperature, 100° to 104°. Five stools, same character. Fed one ounce of the completely peptonized milk by the tube every two hours. Vomited one-half of one feeding. Same treatment.

9th Day.—Temperature, 102° to 103.3°. Stools unchanged. Food not taken so well; not more than one-half ounce could be given; if more attempted, it was regurgitated. Peptonized milk, Mellin's food, plain, malted milk, kumyss and wine-whey were given a trial. Stomach was washed with water at 110° F. After this, one ounce of the whey and kumyss was taken alternately every two hours; vomited the six o'clock feeding of kumyss at 6.30; and in one and one-half hour one drachm of whiskey in one and one-half ounce of completely peptonized milk was forced and retained. The stomach washed and found empty in two hours. The water introduced was withdrawn perfectly clear. The tube feeding repeated in a few minutes and retained. During the night, one ounce was taken by spoon every two hours; one feeding vomited.

10th Day.—Temperature, 101° to 104°. Nine stools; mucus streaked with blood. The spoon feeding of one ounce every two hours continued. At ten o'clock A.M., one ounce of kumyss was given in this manner. Vomited nearly all in one-half hour. Stomach washed; many curds removed. In thirty minutes, one and one-half ounce of kumyss was forced and retained. This was repeated at 1.10 and immediately vomited; the same quantity reintroduced in five minutes, and repeated at 8 and 10 P.M., and *all* retained. During the remainder of night, took one ounce of kumyss and wine-whey alternately every two hours, without vomiting.

11th Day.—Moribund; died in the evening.

Post-mortem examination showed ulcerative colitis and slight congestion of stomach.

CASE III.—*Persistent vomiting, uncontrolled by any means employed.*

A well-nourished female infant, aged four and one-half months, not nursed, was admitted into the Infant Asylum early in July. Vomiting occurred once or twice daily dur-

ing the first nine days, immediately after nursing; the milk vomited unchanged; a greater part of the nursing rejected. The wet-nurse was nursing her own infant, of about the same age, which was healthy. The milk was examined and found normal; microscopic examination negative. The stools remained normal, and the temperature ranged between 98° and 99.5°. The patient was bright and playful, but as there was considerable loss in weight, the nursing was supplemented by two or three feedings daily, of the partially peptonized milk—two ounces given at a time, and allowed to nurse but three minutes. As usual, there was a two-hour interval between feedings. As the breast-milk was vomited when nursed in this manner, and the partially peptonized milk retained, this was given entirely and the nursing discontinued; but the amount given at a feeding had to be reduced to six drachms; if more was taken regurgitation followed. Having noticed many times that the capacity for food was greatly increased by frequent stomach-washing, this measure was employed, but was of no avail; no more than six drachms could be taken and retained. Other foods were tried but with no better success. Stools and temperature normal—on the 13th day of illness, the first rise in temperature occurred —ranged betwen 101° to 102.6°. The stools, heretofore normal, were now green and contained considerable mucus; no vomiting.

14th Day.—Temperature, 101° to 105°. Three green mucous stools; vomited at 10.30 the whole amount given. Baths for temperature *p. r. n.*

Stomach washed at 11; found empty. One-half hour later, fed two drachms of malted milk by spoon, which was vomited at once. After an interval of forty minutes, two drachms of kumyss was given and vomited immediately; later, whiskey largely diluted was rejected every time given. Attempted to feed the malted milk by tube, in the afternoon. Three attempts were made and each time the milk was vomited before the tube could be withdrawn. The temperature at this time was 105°; died two hours later.

Post-mortem examination showed entero-colitis. No stomach lesion to naked eye.

CASE IV.—A delicate, poorly nourished, male infant, aged three months, nursed by mother; began vomiting in April.

1st Day.—Temperature, 99.4°; one normal stool; vomited four times the mother's milk. Upon inquiry we

learned that the patient had been vomiting occasionally during the past week, and that the mother was menstruating; microscopic examination of milk negative. The stomach was washed; the mother ordered to discontinue nursing and feed the baby malted milk by the spoon; during the night one ounce was given every two hours; no vomiting.

2d Day.—Temperature, 99°; one normal stool; one ounce of malted milk given at a feeding, which was vomited; the quantity reduced one-half and vomited—vomiting in all six times, immediately after taking the food, unchanged; no mucus. The patient somewhat prostrated but slept well. During the night, was given six drops of whiskey in two drachms of water, every two hours, and vomited all.

3d Day.—Temperature, 99°. Stools normal; stomach washed at 8 A.M., and partially peptonized milk ordered in teaspoonful doses every half hour. The mother, thinking the child was being starved, nursed it against orders; vomited four times after the peptonized milk as well as after the nursing.

4th Day.—Temperature, 100° to 106.6°.—Five small yellow stools. Stomach washed at 8 A.M. Small quantity of mucus removed; in one-half hour was fed two drachms of peptonized milk by spoon; vomited at once. Later, was given wine-whey, sarco-peptones, and diluted iced-brandy in one drachm doses; all rejected; even vomited one teaspoonful of ice-water. Marked prostration; pulse feeble.

5th Day.—Temperature about 102°; nine stools, composed almost entirely of mucus. Bismuth subnitrate (Squibb's), ten grains every hour ordered, colon irrigated with four pints of tannic-acid solution; temperature, 100°, one ounce of completely peptonized milk was forced every two hours and retained. During night was given one-half ounce of completely peptonized milk by bottle every two hours and vomited nearly all of it; prostration extreme; quite cyanosed.

6th Day.—Temperature ranging 101.8 to 104.5°. Fourteen stools composed almost entirely of mucus; given one ounce of completely peptonized milk by tube every two hours; each feeding contained one and one-half drachms of whiskey; no vomiting. Other treatment unchanged. Baths *p.r.n.*

7th Day.—Temperature, 100° to 100.5°; prostration extreme; pulse almost imperceptible. Tube not used on account of extreme prostration. Feeding by spoon in a

measure successful. About three ounces of peptonized milk with whiskey in small quantities was taken during the next sixteen hours, when death occurred from exhaustion.

The post-mortem examination showed entero-colitis, but no evidence of disease in the stomach.

CASE V.—*A case in which the breast milk and everything given by spoon was vomited repeatedly; no vomiting after forced feeding.*

Delicate female infant nursed by mother; taken sick the first week in July.

1st Day.—Temperature, 100° to 104°. Four green mucous stools; nursed every two hours, the last time at 8 P.M. Vomited nearly the whole nursing; one hour later, at ten o'clock; two teaspoonfuls of barley-water given by spoon was not retained two minutes. In one-half hour, fifteen drops of whiskey was given in two drachms of water, and vomited immediately. At 11.30, nothing having been given in the interval, one and one-half ounce of the completely peptonized milk was forced and retained. During the night rested well; nursed twice by mother without vomiting.

2d Day.—Temperature, 101° to 102°. Two normal stools; nursed every two hours during the morning, three minutes at a time, and vomited three times immediately after nursing; the milk unchanged, contained neither mucus nor curds; the mother's milk examined and found good; microscopic examination negative. During the afternoon one ounce of the completely peptonized milk was given by tube every two hours and retained. During the night was fed once by spoon every two hours and all vomited.

3d Day.—Temperature, 100°; one normal stool; fed by spoon one ounce of the same every two hours; all retained.

4th Day.—Temperature, 102° to 103°; two green mucous stools; during morning fed as yesterday by spoon every two hours; vomited after the eight and ten o'clock feedings. At twelve and two, one ounce of the completely peptonized milk was forced and retained. At four and six o'clock fed the same by spoon; vomited at once after both feedings. At 8 and 10.30, a similar amount was forced and retained. Taken with convulsions at midnight and died twelve hours later; temperature during this time, 104° to 106°.

Post-mortem examination showed entero-colitis and broncho-pneumonia, but no stomach lesion to naked eye.

CASE VI.—*Two attacks of vomiting controlled by forced feeding.*

Healthy female infant, aged eleven months; nursed by mother entirely until nine months old. Since then partly nursed and partly fed on the partially peptonized milk.

Taken sick the second week in July.

1st Day.—Temperature, 102.6°; six green mucous stools: quite restless. The mother was suffering from malaria and the nursing was stopped; microscopic examination of milk negative. Given one drachm of castor oil; ten grains of bismuth subnitrate hourly, and one-fourth of a grain of Dover's powder every two hours.

2d Day.—Temperature, 99° to 101°; ten stools, composed almost entirely of mucus; colon irrigated with two quarts of the tannic acid solution; other treatment unchanged; Mellin's food, plain, the only article of diet which could be taken, was vomited twice in the evening; the vomited matter was composed of mucus and water. The stomach was washed at 9.30, and in one-half hour one-half ounce of the Mellin's food, plain, was given by spoon; this was rejected at once. In one hour one ounce of the Mellin's food, plain, was given by tube, the forced feeding repeated at 11.30, and all retained. During the night not more than one-half ounce could be taken; if more, retching and regurgitation followed.

3d Day.—Temperature, 101° to 103°. The amount given at a feeding, gradually increased until one ounce could be taken by the spoon. One ounce of the Mellin's food, plain, was given alternately with one ounce of wine-whey every two hours, without vomiting. Same treatment continued, with baths for temperature *p. r. n.*

The case developed into one of severe entero-colitis, with temperature ranging between 101°–103.5° for the next ten days. The stools averaged about seven daily, and were composed largely of green mucus, streaked with blood. The diet consisted of wine-whey, sarco-peptones, breast-milk, and peptonized milk. Both salol and resorcine were given a trial; the former was discontinued because the stools became worse while taking it, and the latter on account of the vomiting, which it doubtless started up anew on the 14th day, when both peptonized milk and wine-whey were vomited. The stomach was washed now at 8.30, and in forty minutes the infant was fed one ounce of the completely

peptonized milk by spoon ; vomited the entire amount in twenty minutes. The same was forced at once, and all vomited during the withdrawal of the tube. The tube feeding repeated in three minutes, and all retained. At 11.30 one ounce was given in the same manner, and all was vomited before the tube could be withdrawn. In a few minutes one and one-half ounce was forced and retained.

At 2 A.M. and during remainder of night at two-hour intervals, five drachms was taken from bottle and retained—if more given retching and regurgitation followed.

15th Day.—Temperature, 101° to 103°. Five stools, same character. The quantity taken at a feeding gradually increased from five drachms to one ounce, which was taken every two hours. During the remainder of illness, ten days, a fair amount of nourishment was taken without vomiting. Death from exhaustion due to diarrhœa.

The autopsy showed ulcerative colitis, but no stomach lesion to naked eye.

CASE VII.—A very delicate female infant, aged two months, wet-nursed, taken sick with vomiting in July.

1st Day.—Temperature, 99°; vomited three times; curds and mucus. Stomach washed in the evening, and given one drachm of castor oil. Nursed twice during the night ; no vomiting.

2nd Day.—Temperature, 99°; five thin green stools ; nursed three minutes every two hours, and vomited twice soon after nursing. Milk examined, found good. Microscopic examination negative.

The stomach was washed at 8 o'clock P.M., and one hour later two drachms of wine-whey was given by spoon —vomited immediately. At ten o'clock one ounce of wine-whey was forced, of which one-half was vomited. After an interval of one hour and a half one drachm of whiskey to one ounce of Mellin's food, plain, was forced and retained. Patient very irritable and quite prostrated. During the night one-half ounce of Mellin's food, plain, was fed by spoon every two hours ; this was all that could be taken—if more was given, vomiting and retching followed.

3d Day.—Temperature, 99°; stools normal.

Intense prostration and considerable cyanosis.

Fed one-half ounce of wine-whey, or Mellin's food, plain, by spoon, alternately, every two hours during the morning, and vomited twice the food as taken. The

stomach was washed early in the afternoon, and in one hour one ounce of the malted milk was given by tube; this was repeated in two hours and retained. At 6 o'clock P.M., after an interval of two hours, one ounce of malted milk was fed by spoon and vomited in a few minutes. At eight o'clock one ounce of the completely peptonized milk, fed by spoon, and vomited all in ten minutes. At 9.30 and 11.30 one ounce of the peptonized milk was forced and retained. During the remainder of night one ounce of the peptonized milk was taken by spoon every two hours and retained.

4th Day.—Temperature, 99°; general condition greatly improved; stools normal. One ounce of the completely peptonized milk, fed by spoon, every two hours; no vomiting. The following day was allowed to nurse. No vomiting followed.

The child made an excellent recovery. During the attack, the wet-nurse nursed her own infant, of the same age, which remained well.

CASE VIII.—Delicate, bottle-fed female infant, aged eight months, having recently recovered from an attack of broncho-pneumonia, was taken acutely sick early in July. For about two weeks previous to this time, the food had been taken very poorly. Partially peptonized milk, Mellin's food, wine-whey and malted milk were given a trial; of these, malted milk was taken the best, but not more than one-half ounce could be taken at a feeding; if more given, it was regurgitated. Of course, under this scant diet with one-half drachm of whiskey every two hours, there was rapid failure. The stomach was washed, but it apparently made no difference in the quantity of food taken. Both temperature and stools were normal during this time.

1st Day of Acute Attack.—Temperature normal; seven large, watery stools; vomited twice the malted milk unchanged, which had been given as usual, one-half ounce every two hours. As the entire amount given was vomited as taken, the stomach was *not* washed, and early in the afternoon one ounce of the malted milk was forced; in two hours one and one-half ounce was given in the same manner, both feedings retained—retaining three times as much when forced as when given in the usual way. One ounce was then ordered to be given by bottle every two hours; this was done; no vomiting.

2nd Day.—Temperature normal; seven large, watery stools; put on bismuth subnitrate, ten grains every

hour, with one-fourth of a grain of Dover's powder every two hours; fed as yesterday, and vomited the whole ounce immediately after the ten o'clock feeding. In one hour two and one-half ounces was forced, and, greatly to our surprise, was not vomited. During the afternoon two ounces were given by spoon every two hours and retained. During the remaining seven days of illness, had from seven to nine stools daily. From fourteen to twenty ounces of barley-water, wine-whey and malted milk were taken by hand in twenty-four hours.

During the last three days of illness the temperature ranged very high, and the infant died in convulsions. The post-mortem examination showed slight inflammatory changes in the intestines and broncho-pneumonia. No evidence of disease in stomach to naked eye.

The ages of the cases treated were as follows:

Eleven were under six months; only two over one year.

As regards previous condition and nourishment, six were nursed entirely by the mother, previous to illness; four of these were well-nourished infants, in good health; one was delicate, and one athreptic.

Of these nursing infants the vomiting was permanently controlled in five; in one it was of no benefit; two recovered; two died with ulcerative colitis, one with entero-colitis and broncho-pneumonia, and one from exhaustion.

Two children, both aged two months, were wet-nursed entirely; one was delicate, the other one athreptic. The vomiting was permanently controlled in both—the athreptic died, however, from exhaustion, but the other made an excellent recovery.

Three were partly nursed by mother, and partly fed. Ages ranged from eleven to sixteen months; all healthy. Vomiting permanently controlled in all. One made an excellent recovery, two died with ulcerative colitis.

Two were partly wet-nursed and partly bottle-fed; aged four and one-half and eight months; both healthy. Vomiting greatly improved in one; in the other the treatment was of no service whatever. One died with ulcerative colitis, the other with entero-colitis.

The remaining seven were entirely bottle-fed; four were in fair health, two athreptic, one delicate. Six were

under six months, and one over eighteen months old. The vomiting was permanently controlled in five, improved in one, and no improvement in one. Two recovered; one died with broncho-pneumonia, two with croupous colitis, one from exhaustion, and one with ulcerative colitis.

It will be seen from the foregoing that the vomiting was effectually controlled in fifteen of the twenty cases, and that six recovered.

I am confident that the lives of two of these were saved by the timely use of the tube. In three, with very high temperature, which proved to be due to either croupous or ulcerative colitis, it was of no benefit In three, vomiting ceased to be a symptom, and death followed not earlier than ten days from colitis or broncho-pneumonia, or both.

In two the vomiting was only partially controlled, although three times as much could be taken by tube and retained as could be given in other ways.

In the remaining six fatal cases the vomiting was effectually controlled, thus prolonging life and giving a chance for recovery, which the patient otherwise would not have had.

A feature of great interest, which was brought out by the experiments, lies in the fact that, when but three or four drachms of diluted stimulants or light food could be taken by spoon and retained, three or four times this amount could be forced, not vomited, and the stomach found empty in two hours. This was most noticeable when the spoon or bottle feeding was alternated with the use of the tube. In two cases, in which everything was refused, food given in this manner was retained and digested.

It will be noticed also that the cases in which Gavage was most successful were those of low temperature, in which it did not rise above 103°.

To one who has not washed the infant's stomach many times this method of treatment may seem absurd, as it might be supposed that the tube would cause sufficient irritation or disturbance to produce violent retching and vomiting; but those who have practiced washing of the

stomach on a large scale, and done it properly, will recollect how difficult it is in many cases to make the child vomit, when, on account of thick mucus and large curds, this is desirable. In order to produce vomiting the stomach has to be crowded—filled to overflowing. In the N. Y. Infant Asylum, during the past three years, stomach-washing has been performed not less than fifteen hundred times, and in comparatively few cases does vomiting spontaneously occur during the operation.

Gavage, if properly performed, disturbs the patient but little, and the success in some cases, especially those with very high temperature, certainly depends upon the dexterity with which it is performed. We have frequently introduced one ounce of food into the stomach in this manner—the duration of the whole operation being but fifteen seconds by the watch. In order to do it as rapidly as this, there must be no resistance on the part of the patient. Vomiting of small amounts during the withdrawal of the tube was troublesome in a few. This we found could be obviated to a great extent by *compressing the tube* during the withdrawal as soon as the funnel was emptied. This prevents the small quantity, which always remains in the tube, from escaping into the pharynx, or possibly into the larynx. In addition to this precaution, the tube must be withdrawn rapidly.

The writer thoroughly realizes that twenty cases are not enough to establish a point beyond all possibility of error, but the results obtained show that Gavage is likely to be serviceable:

1st.—In cases of persistent vomiting in infants, not of cerebral origin, after other methods have failed to relieve the patient.

2d.—Under any circumstances when infants can not, or will not, take sufficient nourishment.

3d.—Its application, like stomach washing, must be limited to infancy, since in children over two years the resistance of patients make it almost impossible of application.

DISCUSSION.

DR. HOLT.—It has been my fortune to see a large proportion of these cases that have been reported. I began by being skeptical in regard to the effect of the measure proposed, as many of the gentlemen here are no doubt. I am, however, convinced of its utility. Exactly why it is that food can be fed through a tube and retained when nursing by the bottle or by a spoon causes vomiting, I do not know. It may depend upon the reflex irritation from the pharynx, in cases where the gastric disturbance is not enough to cause vomiting. Certainly it is a fact that with it children can be made to take and digest one and one-half to two ounces of food, when by bottle or spoon one or two drachms are vomited repeatedly. This is worth further investigation. It deserves trial against a symptom than which no other in pediatrics is more troublesome.

DR. O'DWYER.—My experience with feeding by means of the stomach tube, which was sometimes necessary after intubation, before the adoption of the Casselberry method, was very unsatisfactory, as most if not all of the food was usually rejected. Almost all the cases were over the age, which Dr. Kerley says that this method of forced feeding is practicable.

DR. PUTNAM.—Does Dr. O'Dwyer employ the method as described of drawing out the tube quickly?

DR. O'DWYER.—No, I did not. I used the method in older children, and perhaps did not remove the tube quickly enough.

DR. SEIBERT.—Dr. Kerley gave the main points in his paper and asked what should cause the vomiting? It was caused either by the food or by the pathological condition of the stomach and intestines. The cause must be somewhere above the stomach. When we have catarrh of the stomach in an infant, we usually have catarrhal effect upon the oral cavity and pharynx. There is always a certain amount of pharyngeal catarrh, due to the presence of decomposing material and fur upon the tongue. When the tube is put into the stomach, none of this material reaches the stomach. The stomach is practically pure and clean. The sterile food is put in, and then certainly the conditions are much more favorable. In regard to Dr. Koplik's remarks, I do not agree with him. I had a conversation with Prof. Epstein last summer, and my point is, that I am always in the habit, after cleaning out the stomach, of leaving the stomach half full of clear, cold

water, and it is well retained and quickly absorbed. I thank Dr. Kerley for bringing out this therapeutical point so well.

DR. KOPLIK.—I have listened to the paper with great interest, and have followed the histories of the cases. If a child vomits, and this uncontrollably, after the stomach is washed out, we should stop food for a long time after the washing. After washing, you must practically starve the baby for from four to six hours. If the patient is thirsty, you may give a few teaspoonfuls of albumen water. Dr. Epstein, who first used stomach washing, laid great stress on this point. I did not notice in the histories that the little ones had a chance to rest. In this way we might possibly compare the stomach washing and forced feeding to better advantage.

DR. KERLEY.—In answer to Dr. Koplik, in the majority of the babies, if we had waited six or eight hours, we should not have had an opportunity to use the tube or anything else. In most cases one or two hours was allowed to intervene, and then light food, such as wine-whey, was given, sometimes water and whiskey in small quantities.

The mode of action of Gavage in controlling vomiting has been spoken of. I wish in this connection to read a letter from W. Gilman Thompson, M.D., Professor of Physiology in the University Medical College of New York.

"In answer to your inquiry in regard to the possible cause of the interesting phenomena of forced infant feeding discovered by you, I offer the following suggestions:

I. The mechanisms of deglutition and of vomiting are among the most complex nerve reflexes of the body. Deglutition alone employs the action of branches of the facial, trigeminus, stylo-glossus, glosso-pharyngeal, vagus and sympathetic, and in sucking the infant makes special use of the respiratory mechanism, in order to produce a vacuum.

II. The vagus and sympathetic nerves, from their frequent branches to the œsophagus, stomach and heart, can be readily overstimulated through certain branches, with the effect of excitation of others more remote.

III. It is a well-known fact, and one which I have often confirmed on different individuals, that slowly sipping a tumbler of water causes an acceleration of the rate of heart-beat, amounting to fifteen or twenty pulsations per minute. This is due to the effect upon the vagus branches

of frequent excitation in swallowing, for rapidly drinking the entire tumblerful has no such accelerant effect on the heart. The experiment is independent of the temperature of the fluid.

IV. Infants vomit much more readily than adults, with less nausea and respiratory disturbance.

V. It therefore seems possible that under some conditions, especially where gastric indigestion already exists, the vagus and sympathetic stimulation produced by sucking, or taking small swallows of milk from a teaspoon, may become sufficient for it to radiate to the vomiting centre in the medulla and excite emesis.

VI. The nerves of taste and smell readily excite vomiting at times. When milk is swallowed, a coating of it remains upon the tongue and pharnyx, where it forms a favorable nidus for germ culture and fermentation, unless the mouth be thoroughly cleansed. Where stomatitis with gastric dyspepsia exists, this condition would be aggravated, and the nerves of taste or even smell might excite vomiting through the medullary centre. When the milk is put into the stomach through a clean tube, this mouth fermentation cannot occur, and emesis is not excited.

VII. We know how easily vomiting is excited in sensitive subjects by any irritation of the pharynx of a mechanical kind. An infant with gastric dyspepsia and abnormal fermentation begins to suckle. The process lasts for some minutes, and the first mouthfuls swallowed have already begun to ferment in the stomach when the last mouthful, possibly merely by mechanical excitation of the pharynx, causes prompt emesis. In tube feeding the milk is all put down at once, quickly, and the pharnyx is thereafter left at rest, so that no emesis occurs. It seems to me that one or all of these three factors, vagus irritation, bad taste, or mechanical excitation, occuring singly or together, would satisfactorily account for the very instructive and practical observations recorded by you.

Very truly yours,

W. GILMAN THOMPSON, M.D.

FURTHER REPORT ON SUB-MEMBRANOUS LOCAL TREATMENT OF PHARYNGEAL DIPHTHERIA.

BY A. SEIBERT, M.D.,

New York.

IN October of last year I exhibited a syringe devised by me for a sub-membranous local treatment of pharyngeal diphtheria, at a meeting of the staff of the German Hospital and Dispensary in New York. On December 6, 1890, my first report with a full description of this method was published in the *New York Medical Journal*, and at a meeting of the pediatric section of the American Medical Association, held in Washington in May last, I reported on thirty-five cases (ARCHIVES OF PEDIATRICS, June, 1891, p. 428). To-day I can report on fifty more cases, of which four are scarlatinous diphtheria.

This treatment consists in injecting fresh chlorine water (U. S. Pharmacopœia) through the diphtheritic pseudo-membrane into the inflamed mucosa below. I now inject about fifteen drops into each spot, and according to the extension of the process as many as six or eight injections are made in one sitting.

Thirty-seven of these fifty cases occurred in the practice of other physicians, who were kind enough to report them to me for publication; the remaining thirteen were treated by me since May of this year. The gentlemen who favored me with their reports are: Drs. Garrigues and Schlegel, of New York, each with three cases; Dr. H. T. Patrick, of Chicago, with ten cases; Dr. F. H. Bodenius, of Madison, Wis., with ten cases; Dr. Frank Rainie, of Manistique, Mich., with four cases; Dr. L. J. King, of Visalia, California, with six cases, and Dr. J. C. Fritch, of Findlay, Ohio, with one case.

Of these fifty patients five were six to twelve months of age; thirteen were two to four years of age; fifteen were

five to seven years of age; two were nine years of age; ten were ten to thirty years of age.

The injections of chlorine water proved effectual in *checking* the diphtheritic process in forty-two cases; they showed but partial effect in four cases, and had no apparent effect in three cases.

The beneficial effect of this treatment was noticed within three to six hours in seventeen cases; within twelve hours in fourteen cases; within twenty-four hours in ten cases; the time not stated in six cases, and no effect in three cases.

Complete recovery (including disappearance of membranes) after injections occurred in one day in five cases; in two days in seventeen cases; in three to four days in three cases; in five to eight days in eleven cases; not reported in ten cases; died, four cases.

The beneficial effect spoken of consists in a lower temperature, an improved heart action and general better subjective symptoms. Wherever the diphtheritic process is completely checked, all symptoms, with the exception of dysphagia and the visible pseudo-membranes, disappear very suddenly. In Case III., reported by Dr. Patrick, a child of five years, which first had laryngeal diphtheria and was intubated February 26 and the tube removed on March 2, this action of the chlorine water is particularly noticeable, for on the day after the removal of the tube high fever began and a thick membrane formed on the left tonsil. Two injections of a weak solution (0.2 %) of the chlorine water were made, each one being followed by a fall of temperature and pulse from 104° F., and 170 beats to 99° and 128 beats within a few hours. The child was practically well on March 5 and discharged cured soon after. In my last case of fresh pharyngeal diphtheria in a boy aged fourteen years, I made three injections of the officinal aqua chlori in my office at 3 P.M., one into each tonsil and one into the left velum palati. At 8.30 P.M., five and a half hours later, I found the boy in a sound sleep with normal pulse and temperature, the latter having fallen from 102¾° F. Headache, back-

ache and nausea had disappeared, appetite had come. The membranes were all *in situ* still, but the cervical glands had diminished in size. As the pseudo-membranes disappeared the next day, he was discharged cured within twenty-four hours from the time treatment began.

In those cases where an incomplete action of the injections is noticed, the diphtheritic process usually extends over parts of the naso-pharynx not accessible to this syringe. Sometimes the aqua chlori is too old and has lost its strength. The gentlemen who reported these thirty-seven cases all used the weaker solution of 0.2%, which is but one-half as strong as the officinal preparation, and comparatively slower and less effective in its action upon the diphtheritic process. Since I have used the officinal drug and always take fresh-made chlorine water for each visit, my results have even been better than before, inasmuch as the duration of the illness after the injections is shortened even more than before. The little operation should be carefully performed so as to bring the fluid deep enough and to undermine the pseudo-membrane to its full extent.

Of the fifty patients treated four died. Two of these, reported by Dr. Bodenius, children aged seven and seven and a half years, respectively, had septic diphtheria with temperatures of 98.5° and 99° F. when injected; in other words, toxic invasion of the nerve-centres was present, a condition which naturally can never be influenced by any local treatment. I have flatly refused to interfere in several cases seen in this advanced stage of the disease in consultation.

The third fatal case was seen by Dr. Garrigues; simultaneous infection of the mucosa of the whole throat and nose, resulting in death, in spite of injections and other treatment, within five days. Cases of this kind will always occur and will usually terminate fatally. Local treatment cannot influence infected regions not within its reach.

The fourth fatal case, reported by Dr. Schlegel, was one of scarlatinous diphtheria. Tonsils and pharynx

were affected by rapidly spreading necrosis on the seventh day. The injections of chlorine water made then brought this process to a stand-still, the temperature falling from 104° F. to the normal, and the throat clearing two days after this treatment was instituted. Twelve days after the throat was clean this child died, after the opening of a deep-seated cervical abscess. I have treated three cases of fully developed scarlatinous necrosis by daily injections with marked success, the children, aged, two, three and five years respectively, making quick recoveries, and the temperature falling within three hours after each injection. A complete checking of this process at once is of course out of question in this disease.

Of the recovered cases not one developed diphtheritic paralysis.

As to nephritis, nothing was said in these reports, not one of my cases presented this complication, though albuminuria was seen in such as were sick more than three days when treatment began.

Including the thirty-five cases of pharyngeal diphtheria reported before (ARCHIVES OF PEDIATRICS, June, 1891) my collection now comprises eighty-one cases of pharyngeal and four cases of scarlatinous diphtheria treated within the last ten months, with a mortality of six cases, equal to about 7.5 %.

Sub-membranous local injections of chlorine water in pharyngeal diphtheria therefore may be applied with good effect in all cases, where the process is yet accessible to local treatment.

THIRTY-SEVEN CASES OF PHARYNGEAL DIPHTHERIA TREATED BY SUB-MEMBRANOUS INJECTIONS BY OTHER PHYSICIANS.

Cases.	No.	Age.	Duration of Illness when Injected.	Extent of Diphtheria.	How Soon did Injection Act.	Complete Recovery.	Death.	Cause of Death.	Remarks.
Dr. H. F. Patrick, Chicago.	1	3 years	2 days	Tonsils.	Better next day	48 hours.	.....	.....	.....
	2	2½ ..	2 days	Tonsils.	Next day feels well, is about	2 days.	.....	.....	.....
	3	5 ..	6 days	First in larynx, croup, intubation, removal, one day later membrane on tonsils:	In 3 hours, 104-100.°	2 days.	.....	.....	.....
	4	9 ..	2 days	Tonsils, pharynx and palate, nose.	In 24 hours, later no apparent effect, other treatment.	Later, but had diphtheritic paralysis.	.....	.....	.....
	5	4½ ..	3 days	Tonsils, uvula.	In 12 to 24 hours.	2 days.	.....	.....	No other treatment throughout.
	6	4⅛ ..	4 days	Tonsils, later nose, (offensive odor.)	In 12 hours, tonsils free in 3 days, nose after 6 days.	6 days.	.....	.....	.....
	7	19 ..	2 days	Tonsils.	In 12 hours.	24 hours.	.....	.....	.....
	8	6 ..	3 days	Tonsils, pharynx post. and palate.	In 12 hours.	2 days.	.....	.....	.....
	9	22 ..	3 days	Tonsils, post. pharynx, uvula.	In 6 hours.	2 days.	.....	.....	.....
	10	5 ..	7 days	Tonsils and nose.	In 12 hours.	Later under other treatment, though injection acted well.	.....	.....	.....

Cases.	No.	Age.	Duration of Illness when Injected.	Extent of Diphtheria.	How Soon did Injection Act.	Complete Recovery.	Death.	Cause.	Remarks.
Dr. H. J. Garrigues, New York.	1	27 years	4 days	Tonsils and phar.	In 12 hours.	12 hours.	.....	.....	Perfectly well next day.
	2	30 ..	7 hrs.	Tonsils, pharynx, soft palate. Repeated new formations. Very serious case.	1-2°, 2-none, 3-1°,4-marked	1 month. Each injection had a marked effect for good.	.....	.....	Repeated new chills and formations of membrane.
	3	5 ..	2 days	Tonsils, post. wall, finally whole throat.	Apparently no effect.	.....	Fifth day.	Exhaustion.	.....

Cases.	No.	Age.	Duration of Illness when Injected.	Extent of Diphtheria.	How Soon did Temp. Decrease after Injection?	Recovery. How Soon?	Death. How Soon?	Death of What?	Remarks.
Dr. Schlegel, New York.									
Primary Diphtheria	1	4 years	6 days	Both tonsils, uvula and pharynx.	In 24 hours normal temperature 102° F.	2 days.	.....	.....	.....
	2	6 ..	3 days	Tonsils and phar.	Next day, 104-102-98°.	2 days.	.....	.....	.....
Scarlatinal Diphtheria	3	6 ..	7 days	Tonsils and phar., spreading rapidly	104° to normal in 2 days. Diphtheria disappeared	.....	Twelve days after clean, after deep-seated abscess.	throat is child died opening of	....
Dr. J. C. Fritch, Findlay, Ohio.	1	17 ..	2 days	Tonsils.	After 12 hours. 102°-99°.	1 day.	.....	.....	....
Dr. Frank Rainie, Manistique, Mich.	1	18 ..	1 day	Tonsils.	In 24 to 48 hours.	After 48 hours.	.....	.....	Had five cases in one family just before, all lasting long. Treatment: Lactic acid c. c. All recovered. Brother of Case 1 nine years, died the day of illness; sick three days; no injection. (Might have been checked on second day.)
4 cases; 1 family.	2	15 ..	6 days	Tonsils.	In 3 days.	4 days.	.....	.....	
Sick same time. 1st case dead in 3 days (9 years) without injection.	3	9 ..	1 day	Lateral walls of pharynx, palate.	Temperature and pulse better in 6 hours.	2 days.	.....	.....	
	4	6 ..	1 day	Tonsils.	In 6 hours.	3 days.	.....	.....	

Cases.	No.	Age.	Duration of Illness when Injected.	Extent of Diphtheria.	How Soon did Injection Act?	Complete Recovery.	Death.	Cause of Death.	Remarks.
Dr. F. H. Bodenius, Madison, Wis.	1	1 year	.....	Tonsils.	In 5 hours, 104-100°.	All discharged cured within five to nine days.	.....	.....	.....
	2	7 ..	.....	Tonsils and uvula.	In 5 hours, 102-99½°.		.....	.....	.....
	3	6½ ..	.....	Tonsils and uvula.	In 5 hours, 103½-100°.		.....	.....	.....
	4	11 mos	.....	Lateral walls of pharynx.	In 5 hours, 103⅘-101.		.....	.....	.....
	5	9 ..	.....	Tonsils.	In 5 hours, 102-100°.		.....	.....	.....
	6	7 years	.....	Pharynx, tonsils and uvula.	In 5 hours, 104-101⅗°.		.....	.....	.....
	7	6 mos	.....	Tonsils.	In 5 hours, 103-100⅗°.		.....	.....	.....
	8	8 ..	.....	Tonsils and uvula.	In 6 hours, 102-99⅘°.		.....	.....	.....
	9	7 years	.....	Septic diphtheria.	None.	.....	.....	Myocarditis.	.....
	10	7½ ..	.....	Septic diphtheria.	None.	.....	.....	Nephritis.	.....
Dr. L. J. King, Visalia, Cal.	1	.....	.....	.....	.....	All recovered. Satisfaction.	.....	.....	.....
	2	.....	.....	.....	.....		.....	.....	.....
	3	.....	.....	.....	.....		.....	.....	.....
	4	.....	.....	.....	.....		.....	.....	.....
	5	.....	.....	.....	.....		.....	.....	.....
	6	.....	.....	.....	.....		.....	.....	.....

DISCUSSION.

DR. KOPLIK.—I think Dr. Seibert's report is a bacteriological experiment as applied in therapeutics. Dr. Seibert is very modest in his claims. He does not claim to cure every case. I remember the time when diphtheria locally was treated with the actual cautery and the strong stick of silver nitrate. Later we were told to leave the membrane alone. Now there is a movement in the opposite direction. We could talk all day on the local treatment of diphtheria, but we must remember one thing, *that the severity of any disease varies with the epidemic.* Whether the lesion be local or constitutional, or both, the severity and mortality vary, and in no disease is this so marked as in diphtheria. We cannot forget that diphtheria is also a disease of the greatest constitutional severity. Long before we are called to the bedside, in some cases even with a limited local lesion, the die is cast and we can often say as soon as we see the patient that he is going to die. In one case I was called on the third day of the child's illness, and though the child lived fourteen days I made the prognosis at that first visit that the disease would be fatal, the poison having fairly overpowered the constitution from the start. I do not say that we should leave the membrane alone or treat it harshly. We must remember in our treatment that the tonsil is a lymphatic gland or node with one side exposed. The injection reaches only locally where you penetrate. In marked cases there is extensive cervical adenitis. It seems to me that in some of these cases, even after the membrane disappears, the patient is doomed. The system is already poisoned. You look at the heart, kidneys, and other tissues post-mortem, and you see that recovery would have been impossible. We have spray and treat locally and make bacteriological experiments, but we should not forget the anatomy of the part nor the constitutional effects of the disease. Dr. Seibert may stand on this ground. Dr. Fraenkel and others are working on a constitutional basis. They are working to obtain effects that drugs do in syphilis, and are sanguine in expecting to see bacteriological experiments performed right into the circulation, and that thus the local lesion may be affected and cured through a constitutional treatment after the system has been fortified by something which the disease is powerless against. Look at laryngeal diphtheria. Nothing is done except to make the patient breathe. The local lesion is not treated, and in spite of this we have recover-

ies varying from 24 per cent. upward. The day will come when we will intubate, and then we will do something else that will lessen the time of recovery, cure the local lesion and save the patient from complications.

DR. NORTHRUP.—I would like to know upon what Dr. Koplik bases his statement that this is correct bacteriological treatment?

DR. KOPLIK.—I do not mean to convey the idea that this is absolutely correct treatment. I mean that it is simply an attempt to affect a case by local treatment with antimycotic injections. I doubt whether this attempt will be the best.

DR. NORTHRUP.—How deep do the bacteria go? Dr. Seibert makes deep injections into the tonsils. Now, as to the glands of the neck, are the deep injections to have any effect upon them?

DR. KOPLIK.—The bacteria of diphtheria are not found in the deep tissues. The effects of their action are found in the tissues and internal organs. How deep Dr. Seibert means to go and affect the tissues is for him to answer. I think the injections stay where they are put. I do not think it is possible to affect the deeper lymphatic. If we could do this we might annihilate or counteract some of the poison which invades the lymph glands and kills the patient.

DR. SMITH.—Will Dr. Seibert state how much of the antiseptic he employs?

DR. SEIBERT.—Fifteen drops for each injection.

DR. SMITH.—Do the points extend deeply enough to reach the vessels? Is there any hæmorrhage?

DR. SEIBERT.—The points are supposed to reach the lower layer of the inflamed mucosa. Occasionally we have slight hæmorrhage of a few drops. The points will certainly reach the capillaries where the pseudo-membrane is not thick.

DR. SMITH.—It seems to me that the fact that the blood vessels are lacerated and opened by this operation is an objection to such treatment, because the toxic albuminoid which is generated in the pseudo-membrane and upon the inflamed surface, by the action of the Klebs-Loeffler bacillus, and which, taken up by the blood-vessels and lymphatics, cause systemic infection, could enter the system readily through the lacerated capillaries, would not also these punctures be likely to increase the inflammation?

DR. NORTHRUP.—I do not want to get up every moment, but I am interested greatly in this matter. I am willing to accept the facts that Dr. Seibert gives and approve so far as I understand the operation; but we have also definite facts in bacteriology. When Dr. Seibert introduces deep antiseptic treatment, is he trying to counteract the bacteria deep in? Let us try to get at some of the facts first and not discuss explanations which are most confusing. There are ptomaines or toxines or what-not, but the *bacteria are limited to the superficial surface.*

DR. HOLT.—I would like to ask the age of these patients?

DR. SEIBERT.—Five of them were from six to twelve months of age, thirteen from two to four years.

DR. HOLT.—My experience may be unfortunate. I have come to the conclusion that the drug treatment of diphtheria is a failure. Of course the results vary with the severity of the epidemic. In one institution in ninety-one consecutive cases, nearly all of which were under two years of age, the mortality was 60 per cent. I am not encouraged with that experience or any other to persevere with the drug treatment, and therefore I think this treatment of Dr. Seibert's is in the right direction. Diphtheria, I believe, is first a local, then a constitutional disease. It is rational as in septicæmia to attack the local disease vigorously. If Dr. Seibert puts into the capillary vessels an antiseptic solution, it will dispose of the bacilli which may chance to enter at the same time. The results reported justify us in trying this method carefully. Parenchymatous injections into the tonsils have been used for twenty years and successfully. I think we are driven to local treatment if we wish to influence the progress of the disease in its early stage. Gargles and sprays are often used with the idea that this is germicidal treatment. This is a mistake; they may be of great value in securing cleanliness, but they are not to be considered germicidal. We have not removed the dangers of the patient from toxæmia. The great obstacle to the treatment proposed is the difficulty in making the injections in young and unruly children. Two people would seem to be needed besides the operator—one to hold the patient's body and one to steady his head. This, however, is not a vital objection if the operation is as successful as Dr. Seibert believes it to be. The question is not how much of an operation is it, but how much does it benefit the child? For one I shall give it a thorough trial.

DR. ROTCH.—I should like to ask the proportion of cases in which the tonsils were affected and in which there was no membrane on them. It makes a great difference what parts are covered by the diphtheritic membrane —whether the tonsils of the naso-pharynx, the later being much more richly supplied with lymphatics than are the tonsils, septic absorption takes place far more readily and the disease at once assumes a more fatal type.

DR. SEIBERT.—I have a chart showing this and other details. In but three cases the tonsils alone were affected. So that it can be left out of the question that we had to do with a benign affection of the tonsils. The Loeffler bacillus was found in over thirty of the cases ; in twenty I did not find it. In many I did not look. I make it a point to examine the membrane whenever practicable. It is easy to find the bacillus, and it excludes the possibility that we have to do with a non-diphtheritic trouble.

DR. ROTCH.—I did not in any way question that they were true cases of diphtheria, but merely wished to know the location and extent of the membrane.

DR. SEIBERT.—In nearly all the cases the tonsils were affected, but the disease was confined to them in only three of my own cases. Twenty-one of twenty-four cases reported by others showed other parts of the pharynx affected besides the tonsils.

DR. KOPLIK.—I wish to ask whether the Doctor has made any examination as to the diffusion of the liquid in and around where the injections were made ? As I understand him, he gets under the membrane and then injects the fluid. How widely underneath the membrane are the effects carried and the albuminoid substances affected ?

DR. PUTNAM.—Were there only three cases in which the disease was limited to the tonsil ? How many had extensive lesions ?

DR. SEIBERT.—In more than one-half of the cases that I treated, and crediting to the histories of others, the disease had invaded the visible pharynx ; whenever the naso-pharynx was also affected the effect of the injections upon the visible parts of the diseased mucosa was always noticeable.

DR. PUTNAM.—Have you seen, the day following, extension of the membrane in other directions ?

DR. SEIBERT.—I have seen an extension in all cases in which I was not thorough enough—where I was too timid and made only one injection where I should have made two. The chlorine water acts only where it is brought.

How far it goes I do not know. In rabbits and guinea pigs, which I used in my experiments, the injections caused a swelling lasting from two to three days, gradually passing away.

DR. PUTNAM.—Was there any evidence that the extension was due to the injection?

DR. SEIBERT.—Never. In most of my cases I had œdema of the pharynx, a succulent uvula and an angry appearance of the pharynx, usually seen in genuine diphtheria. Six hours after the injection the parts around the injected spot look pale, and the œdema disappears usually within twenty-four hours. I have used the same syringe in all cases, all winter, and had chlorine water injected into my own tonsils, and no ill effects ever occurred. Chlorine water will kill anything on the instrument in a few seconds, and is perfectly safe. A niece of mine, aged five years, was treated five days for follicular pharyngitis before I saw her. I found diphtheria of the soft palate, the uvula, the posterior and lateral walls of the pharynx down to the larynx, and of the tonsils. The child had cold extremities, a miserably fast pulse, and a temperature of 99° in the rectum. I did not think the child would live another twenty-four hours. In spite of this opinion the parents insisted on my using injections. I made seven at once. The next day the temperature was 101½°; pulse 120, and strong. Three days later the child was absolutely well. If my own child was affected to-morrow, I would not be afraid, provided I could reach the diphtheritic spot with my syringe. Where does the chlorine water go? If diphtheria is a local disease, and if the bacilli of Loeffler are found under the pseudo-membrane, then we must bring our drug to that spot. We have bacilli in the mouth, but to produce diphtheria it is absolutely essential that they *enter the mucosa.* This is Oertel's teaching and also Heubner's. There can be no diphtheria unless the bacilli reach the capillaries of the mucosa. The bacilli enter into the walls of the vessels and cause the inflammation. The ptomaines do not cause the inflammation, or we should have diphtheritic inflammations following the absorption of the diphtheritic tox-albumen, instead of the well-known paralysis. It is *not* intended to make *deep* injections, but to bring the needle-points and the chlorine water *into, and immediately below, the inflamed mucosa.*

Whatever the action of the procedure may be, it will be well to make a trial, as in stomach-washing. When I introduced stomach-washing in infants here, and stated

that with Epstein's method we could check nausea in cholera infantum, provided that the central nervous system was not invaded by the milk poison, very few believed what I said, and you can read in the "ARCHIVES" of a young enthusiast going around with a stomach tube sticking conceitedly out of his coat-tail pocket, and I presume I am the man referred to. I think I am understood by all. Diphtheria is first a local disease of the mucosa. How did the mucosa become diseased? The bacilli must penetrate the epithelial layers of the mucosa so as to get down to the lower stratum containing blood-vessels, irritating these and causing exudation of serum into the epithelial layers, thus forming the pseudo-membrane. The tox-albumen causing diphtheritic paralysis is a product of the action of the Loeffler bacilli upon tissue-albumen. As this is not present upon the surface of the mucosa, it is evident that the bacilli must *enter* the tissue to come into close contact with this albumen necessary for the toxin. Where are the bacilli before the membrane forms? They certainly have entered the mucosa and *caused* the pseudo-membrane to form. First comes the intermingling of sub-mucosa and bacilli, then the result, the pseudo-membrane. Therefore our treatment must attack this invaded region. Whether chlorine water injected here destroys the bacilli directly, or so changes the tissue as to stop bacterial growth, is immaterial; certain it is that it acts promptly and germicidal. Chlorine water only acts *locally*, as the chlorine immediately combines with surrounding tissue. Sublimate and carbolic acid would be readily absorbed and might injure. Intra-tonsillar injections of a 3 per cent. solution of carbolic acid have been made by Heubner in scarlatina alone for the last ten years. He has had good results. My injections are not deep, only deep enough to reach the lower stratum of the invaded mucosa.* Sub-membranous injections in diphtheria were first devised by me. In fifty cases I only used a gag in an unruly boy of fourteen years, and then only for the first injection. The smaller the child, the easier it is to make them obey. The mother holds the child, and any one can hold the head; the tongue is pressed down by a spoon, the syringe is passed over the spoon until the points are in position; these are then pressed through the pseudo-membrane as far as necessary, then the spoon is dropped, and the injection is made

* The needle-points are now made of two sizes, the one one-eighth and the other one-sixteenth of an inch in length.

slowly. I have never had any trouble, nor have I been dismissed from any family on the ground of the severity of the treatment.

DR. FRUITNIGHT.—How frequently were the injections made?

DR. SEIBERT.—If you make the first injections thoroughly, you may not have to make any more in *primary* diphtheria. In my niece I used the syringe seven times during the first visit, and no more. Of course I have other treatment besides this. I treat the child and not the symptom. As a rule one injection under each pseudomembrane is enough. If the temperature does not fall, the injection was not right in quantity or quality, or you have not reached the right spot. Eight times at one sitting is certainly enough. Two injections on three successive days was the extreme number made in a case of pharyngeal diphtheria. Wherever the toxic symptoms are so far developed as to let them point to an early heart paralysis, I will abstain from all local treatment, for this is purely local treatment, and can only *prevent* general symptoms if employed early.

DR. ROTCH.—This treatment, though not in accordance with our knowledge of the anatomy and physiology of the tonsil, has apparently proved successful, clinically, in Dr. Seibert's hands. I would like to ask if chlorine water is undoubtedly antiseptic?

DR. SEIBERT.—Geppert, in Bonn, has made investigations as to the properties of chlorine water. Schmidt-Rimpler has within a few weeks published an article that corroborates the statement that chlorine water is more germicidal in its action than other antiseptics, and is one of the best we have.

MANIFESTATIONS OF "LA GRIPPE" IN CHILDREN.

BY CHAS. WARRINGTON EARLE, M.D.,

Chicago.

WHEN the history of the present epidemic (1891-'92) is written, three consecutive yearly visitations of La Grippe will have been recorded. The occurrence of this disease with such frequency, may in my judgment be considered, and spoken of as a national calamity. The troubles, anxieties and deaths among our families—in addition to the increased labor which nearly prostrated our profession, and in many cases was the direct cause of death of some of its members—to say nothing of the sequelæ which have followed and which will continue to follow, all unite to make it an epidemic much to be dreaded.

The disease "*La Grippe*" or influenza, has been fully recognized since the sixteenth century.* Inasmuch as it has been described in our own country for nearly two hundred and fifty years it is a matter of surprise that the literature is not more exhaustive, especially in our standard works.

* The first epidemic in America, according to Webster, took place in 1647.

Wilson in his treatise on "*Continued Fevers*," has an excellent article on *La Grippe*, but says in regard to some epidemics that children remain relatively exempt from its seizures.

This may be true of some epidemics, but it certainly was not the case, during the winter and spring of 1890, and 1891 (nor thus far in 1892) children having suffered to a very great degree.

At the commencement I may say that I am at a loss to account for the paucity of facts which the average practitioner seems now able to offer in regard to the cases so recently under his observation. Many of the details, even the number of cases and the more striking of the constant phenomena, have, seemingly, been lightly impressed upon many of us. And yet I know, and will allow for this largely from the fact that we were so busy that it was absolutely impossible to keep the records which we ordinarily do. That close observation and fine systematization which is permitted by leisure moments—moments of calm thought—does not come with times of intense mental and physical stress—almost exhaustion.

General Considerations.—This disease is, without doubt, a general infection of the entire body. It takes possession of the system with great rapidity, indeed it has been called by some writers "lightning catarrh." It involves not only the respiratory tract and digestive apparatus—where we see the catarrhal manifestations most prominently—but the nervous system is also taken possession of with quite equal rapidity.

It has been shown that previous to and at the beginning of the recent great epidemic of influenza—which was said to have taken its start from St. Petersburg—the prevailing wind was unusual, and that its direction was from the northeast, from the cold, desolate and exile-populated region of Northern Siberia.

It has also been shown, from the admirable and painstaking reports under the care of the Michigan State Board of Health, that previous to and during the commencement

of the epidemic in that State, there was an unusual direction of the wind, notably from the *northeast*, and that, also, there was a marked increase in the amount of atmospheric ozone.

Again, it has been recorded by an able English observer, who carefully traced the course of the spread of the disease in its late appearance, that it clearly depended upon the lines and channels of human travel—that after its marked occurrence in St. Petersburg it worked its way eastward, the prevailing northeast wind possibly exercising some influence, following mainly, and very apparently, the international railway lines and great highways, passing in this way, and successively, through Germany, France, and other of the western continental nations of Europe, thence to England, across the Atlantic to the United States, progressively across the continent from east to west, thence across the Pacific to Japan and China. It then appeared to double on itself, taking a backward course, though with boundaries much less distinct.

Although the point of commencement of the great epidemic of 1889-'90 has been somewhat indefinitely given as being "east," and probably northeast of St. Petersburg, and although it has been synonomously termed "Russian Influenza" from its supposed origin in the Russian provinces, yet later investigations go to show that a peculiar disease having the characteristics of influenza prevailed throughout portions of China—and particularly in Hong Kong —in 1888, and that from thence it traveled across Siberia and first attracted general notice at the Russian capitol. This being the case it only adds to the remarkable nature of the disease, and this last epidemic, for it completes the evidence of the circumterraneous march of the affection. The course of the disease was westward constantly from its starting point in China until China was reached again after encircling the earth. The Russians called the disease "Chinese Influenza"; the people of the west termed it "Russian Influenza"; while later on, when it reached China again covered with renown yet the object of horror,

its name was once more changed to "Japanese Influenza," for its direction was from the east—from Japan. Dr. Joseph Cantlie, of Hong Kong says: "I contend that influenza is endemic in China, and that the prevalence of the disease in Hong Kong in September and October, 1888, was the start of the recent epidemic elsewhere."

In considering the course of the disease longitudinally there appeared to be attraction in the direction of the equator. This was observed in both the northern and southern hemispheres.

The report to the British Local Government Board, by its Medical Officer, clearly shows that the spread of the influenza depended upon human intercourse—that the disease spread no faster "than human beings, parcels, or letters could travel."

The commencement of the epidemic of 1891 was about middle of February, although a few cases occurred as early as January 1st. In my judgment the influence of this epidemic was felt by many for weeks before its manifestations were grave enough for us to diagnosticate it as a separate disease. There was a sense of weariness—a depression—a tendency to suffer pain in the back and extremities that was before this unknown. The height of the scourge was reached about the 20th of February, and it gradually diminished until the epidemic can be said to have ceased about the last of March. Its period of greatest intensity may thus be defined as from four to six weeks, which is about the usual time given in historical notices descriptive of this disease.

Age.—No age has been exempt from the ravages of this infection, and if children have been spared in other epidemics they did not escape the present one. Nursing babies at four months of age felt its influence, while a large number between one and three years suffered. Quite as many under the five-year mark were affected as between five and ten years.

One observer says: "The younger the child the more severe the grip."

Dr. P. Strassman *(Zeitschrift f. Geburtshülfe und Gynäkolog.*, Abst in *Archiv für Kinderheilkunde*, 1891, p. 132*)* says :—"In January 1890 infants became sick with symptoms of influenza. Child became uneasy, clear green nasal secretion, rapid respiration, voice hoarse, cough frequent. Also digestive disturbances in the form of either constipation or diarrhœa. In rectum the temperature was frequently found below normal—95°, 92°, 88.1°. All showed loss of weight. Youngest was three, oldest fourteen days old. One died from pneumonia. In two there was mild conjunctivitis, and in three there was an exacerbation of previous conjunctival inflammation."

CASES.—Some of the peculiar manifestations of this disease in children can best be understood and studied by narrating a few cases.

CASE I.—Baby G. age ten months. Sick two weeks before I was called with a complaint diagnosticated by an "irregular" as "teething." Physical examination revealed a slight pneumonia of the left lung, and in two days a general capillary bronchitis developed in both of the lungs. The bowels were loose, and there was some vomiting. Four days before death the child became restless and delirious, and would toss its head about, bite its hands, and was generally irritable. Later it became comatose, and death took place about one month after the commencement of the disease.

CASE II.—Baby McC. age ten months. When first seen this child appeared to have an ordinary bronchitis and to be in no danger, but in three days an inflammation of the small tubes had developed with exceedingly severe symptoms. There was the usual rapid breathing, more or less pain, and a temperature which repeatedly arose to 105 degrees F. Very early in the disease convulsions were present, which were not followed by symptons particularly referable to the brain ; but four days before its death—which took place at the end of the second week—all the symptoms of meningitis, that is, convulsions, paresis, changeable pupils, contractions and finaly coma, were present.

CASE III.—Baby H., age, twelve months ; bottle-fed and naturally delicate. Was called to see him on March

23, and found evidence of headache ; a condition of lassitude and weariness ; wanted to sleep and be let alone ; was nervous and suffering from exhaustion. These symptoms continued for fifteen days, when the child seemed actually better and the visit on the sixteenth day was omitted. The following day, however, a bronchitis, which may be said to have been "smouldering," suddenly increased in severity and the nervousness became more marked. The child became drowsy, was aroused with difficulty, and on the twenty-first day of the disease death resulted. Three days before death the ears began to discharge, and throughout the early days of the sickness there were symptoms of gastro-intestinal complication, there being vomiting and from six to ten bowel movements each day.

CASE IV.—Baby C., age, ten months. Pneumonia present from the first visit, and death in five days. In addition to the slight pneumonia there developed on the second day a capillary bronchitis throughout both lungs, with great dyspnœa, temperature 104–105° F., and a pulse from 140–150°. There were great nervous disturbances during the course of the entire illness, the child being constantly agitated, wanting to bite its hands, and was delirious to the last, though not comatose.

CASE V.—Baby D., age, eleven months, and nursing its mother. This child had a slight attack of bronchitis early in the epidemic, but recovered fairly after my third or fourth visit. In the course of a few days I was recalled and found trouble from a severe gastro-intestinal disturbance, the little one refusing its food and having from ten to twelve bowel movements daily. It refused to nurse its mother during its sickness, and was nourished upon the white of egg, brandy and a little rice-water. Very early in the disease this child would cry when it was moved, although the greatest care was exercised. A slight bronchitis took place during this relapse, but did not become serious. Nervous symptoms made their appearance and the patient was apparently sleepy at all times. It would remain with its eyes wide open for twenty-four hours at a time, but there were no convulsions, and in the course of three weeks this child made an excellent recovery, and is in a good condition of health at the present time.

CASE VI.—Baby J., age, one-and-a-half years. This patient was seen in consultation with one of the oldest

and most eminent practitioners of Chicago. The child had passed through a mild bronchitis, its nutrition had been much impaired, and it was, at the date of my first visit, drowsy and crying out in a manner resembling in some degree what is called the "hydrocephalic cry." The pupils were equal, however, and responded to light throughout the entire course of the affection. This child was watched with the utmost solicitude, as we hardly dared to hope that meningitis would not develop. None of the symptoms which are usually prevalent in this last-named disease made their appearance however, and after two or three weeks of particular attention to its nutrition, and such stimulation as we felt warranted in giving, it made an excellent recovery, and is fat and rosy to-day.

CASE VII.—Baby M., age, four years. This child had a very unusually developed nervous system, and upon the slightest exposure always had a high temperature. During the first days of his sickness the nervous symptoms predominated, but these, however, gave way, and for three weeks there were present in his lungs all the evidences of a general diffused bronchitis of the medium-sized tubes, with some indications of localization in the lower right lung. He lost flesh to an alarming extent; had a temperature which persisted for a long time; refused food for many days, but finally convalesced and is now going on as well as can be desired. I was particularly fearful of empyæma in this case, and made repeated examinations lest I should be mistaken.

CASE VIII.—Baby P., age, two years. Was called to see this child by a messenger who stated to me that the patient was in the midst of cerebro-spinal meningitis. I found the little one drowsy, vomiting, refusing all food, and with a considerable diarrhœa. In this case it seemed as if a meningitis would certainly develop with the usual fatal termination, but after watching it closely for eight or ten days, and with most assiduous nursing on the part of the parents, its convalescence commenced. This child was fed upon artificial food previous to its illness, and during its convalescence it was impossible to furnish it anything else. The parents were instructed how to sterilize food properly, and, with the exception of one or two very slight attacks of gastro-intestinal disturbance through the summer, it has progressed favorably and promises to become a perfectly well and healthy child.

Etiology.—The hypothetical causes of the disease have been classified as follows: (1). Poisonous changes in the air. (2). Contagion. (3). Products of local conditions and general influences.

Concerning the bacteriology of influenza little has been written. Germs of different character have been found in the muco-purulent discharges from the respiratory passages, but it is universally acknowledged that the particular bacterium has not, up to this time, been isolated. (It is just currently reported that Pfeiffer, of Berlin, has isolated the evident bacillus of influenza, and been successful in inoculations. The bacillus is said to be the most minute yet discovered.) It is, therefore, along this line that we should work in future epidemics to discover, if possible, the absolute cause of this disease.

Whether this disease is contagious or not is a question which at present must remain undecided, although there are many facts which go to show that it does, at least, possess this quality mildly. Dr. Herrick, of Chicago, gives the following observations:

"Father, mother, and two children sick in same week. Four children in one family sick during same week. Father, mother. and child sick during same week. Mother, infant one week old, and three children sick the same week. Three children sick at same time. Two children and older brother sick during same week. Child sick, then mother, then friend in same house who helped care for the child. Two children and mother sick in same week. Mother sick, two days later child nine years of age sick."

In many families nursing children had the disease two or three days after the mother.

Some writers deny any contagious nature whatever, and Hirsh makes a point that it does not spread in our day (the railroad age) more rapidly than it did one hundred years ago. The disease has also been known to rage in limited regions, and has refused to spread in spite of repeated communications. Opposed to this it has been known to take place as an epidemic on an island—where

the disease was unknown—after the arrival of a vessel; and in many cases every member of the same family has been afflicted with the disease at the same time, or within a few days.

Filatow (Arch. für Kinderheilkunde, Vol. V., p. 357), has written fully concerning the history and symptoms of the disease under consideration, and Otto Seifert (Samml. klin. Voetr., No. 240) has investigated its bacteriological history in a very thorough manner. I quote freely, particularly from the last author.

"If this disease is caused by a poison of organic nature, one might succeed in finding it in the blood or secretions.

"As long as the fever was at its height, the nasal and bronchial secretions were tenacious and mucous. In the sputum there were imbedded grayish-white gelatinous clots. As soon as the fever decreased and the subjective symptoms began to abate, the character of the secretions changed. Nasal mucus and sputum began to grow more liquid and muco-purulent, diminished in quantity as the patient improved, and at last became normal. To this difference in quantity and quality corresponded what was found by means of the microscope.

"*Preparation.*—Some of the most tenacious and transparent portion of the sputum and some of the gelatinous clots were colored with aniline dye in the usual manner. First, they were spread between cover-glasses, dried over a spirit lamp and put in methyl-violet solution, being left there for an hour. After washing with alcohol, and being dried once more, they were put into Canada balsam.

"*Under the Microscope.*—There are only a few cells, the tenacious mucus is drawn out in long, thin strings. Imbedded in these strings or beside them are an uncommonly large number of micrococci, measuring 1.5 *u* to 2 *u* in length, and 1 *u* in width. (*u* equals one one-thousandth of a millimetre, known as micro-millimetre, and is 1-25000th of an inch). Not in a single place are they in any relation with the pus cells, lying neither in nor upon them. As far as the arrangement of the cocci are concerned they lie mostly in long

chains, rarely by twos (diplococci) very frequently alone (monococci). The examination of the tenacious mucus from the nose showed the same result.

"It was now determined to (1). Examine the secretions of the respiratory passages in all of our influenza patients. (2). To make 'control' examinations. (3). To examine whether the blood contained micrococci. (4). To attempt vaccinations on animals.

Result.—I. We succeeded in every case to demonstrate the existence of micrococci in the nasal mucus and in the sputa. In staining, we did not limit ourselves to methyl-violet, but also tried other aniline dyes, whereby was shown that fuchsin was less good than vesuvin, and that double-staining did not succeed at all. Thus it was demonstrated that this micrococcus belongs to those forms which do not retain their first color. All other staining was done with methyl-violet. The secretions did not always contain an equal number of cocci, these latter were most numerous in the gelatinous clots, but also very numerous in the tenacious, transparent portions of mucus. As soon as the secretion assumed a muco-purulent character and increased in number of cells the number of cocci decreased remarkably, and in those portions which showed the greatest number of cells, there were just a few cocci scattered between the rows of cells. In the conjunctival sac we did not find any cocci.

"2. *Control examinations* :—The nasal secretion of three persons, suffering from an ordinary cold, were examined, and of two persons having contracted a cold in consequence of measles. In *none* of these cases have we been able to find micrococci as were described above for influenza. Also the sputa of six persons suffering from simple bronchitis were examined, as well as the sputa of those two persons sick of measles. No micrococci were found.

"3. The blood of the influenza patients was most carefully examined. No micrococci were found.

"4. The fresh nasal and bronchial secretion of an influenza patient was used for the vaccination of two rabbits, into the nasal cavity and the oral cavity respectively. On

the following day there was no change visible in either cavity of the infected animals. There was a slight increase in temperature. On the third day the conjunctiva of one of the animals were reddened, otherwise no change. On the fourth day a very slight increase in temperature; on the fifth day both animals were well again."

It is the question now, whether, on the ground of the above examinations, we are justified in believing in a living micro-organism as responsible for the occurrence of influenza. That absolute proof is in our possession showing that the cocci found in the nasal and bronchial secretions of the patients forms the only and direct cause of the disease, cannot be claimed as certainly demonstrated. Although the "control" examinations point in this direction, there have yet been no attempts made at cultures, and no vaccinations tried with all facilities insuring success.

Despite this, our author believes influenza to be caused by the above-described cocci, which are carried by the air to the respiratory passages, there finding a suitable soil for their growth and development.

Conditions of life of these low organisms:—The cocci secreted with the nasal and bronchial mucus, and in this condition being dried, are taken by the air and are carried rapidly from place to place. It is in this manner that the infection is transmitted through the medium of the atmosphere.

Life of the cocci:—If we believe them to be the specific cause of influenza they might be called *influenza cocci*, and we have to imagine that they increase in numbers on the mucous membrane of the respiratory passages, and by so doing produce the usual symptoms of the disease. It is not necessary that the mucous membrane should have been prepared for the reception of the cocci, for they spread on previously perfectly healthy membrane. As an explanation of the fact that not all persons are attacked by the disease, we assume a special individual predisposition within physiological limits. An analysis of the clinical phenomena shows that the intensity of the disease

is not dependent upon the number of the cocci. The local symptoms alone may be caused by the number and spreading of the cocci, which, by increasing in great numbers on the respiratory mucous membranes, cause an inflammation which will be noticed as an objective symptom. After a certain time there will be an accumulation, and an increased excretion (proliferation) of lymph cells in the endangered portion, and, in consequence of this reaction the changes in the tissues are going on more energetically, and during this competition between cells and bacteria, the former gains the victory (this is the Metschnikoffian doctrine—the hypothesis of phagocytosis).

Theories: 1st. The cocci may be simply flooded away by the increased excretion of cells, that is, by the more profuse and muco-purulent secretion.

2. They may be killed by certain chemical products of the cells. If we credit the cells with the production of certain chemical substances, we may attribute the same property to the cocci, the difference being that the product of the cocci is toxic in nature. The considerable amount of backache and pain in the limbs are assuredly not caused by the moderate fever, or they would disappear rapidly after the fever is gone. One of the symptoms that may be acknowledged as a special peculiarity of influenza is the slow convalescence, and the fact that the patient will complain of backache and pain in the limbs for weeks after the disappearance of the fever; which goes to show that the poisonous products of the cocci can only be slowly eliminated from the system.

The cocci usually remain localized on the respiratory mucous membrane, as shown in the negative results of the blood examination, and by the absence of swelling of the spleen. It is believed that in a few single severe cases the cocci may pass into the blood. This may be concluded more easily from the observations of Lenffl, who has shown that swelling of the spleen has taken place in severe cases in some former epidemics, and that the disease has raged so severely as to cause sudden death.

Incubation and prodromes.—The incubation seems to occupy only two or three days, and in this respect resembles scarlet fever. After the disease is once introduced into the family only a short time elapses before it develops in those not previously afflicted. The prodromic period is not well marked, although slight catarrhal difficulties, not sufficiently severe to be much noticed, with some pain in the head and extremities, are the symptoms which become manifest at first, and are only remembered after the disease has fully set in. The very slight symptoms which were noticed in many—such as weariness, mild catarrh, either of the respiratory or digestive tract—were in all probability rudimentary, or abortive, attacks of influenza.

According to Unger a catarrh always developed, that is, symptoms referable to the mucous membranes of the respiratory system were always present. I cannot say as much from my experience. Some profound cases have taken place, in which the nervous system has been the only part of the body involved, the debility, headache, and terrible pain in the body and limbs being the only symptoms, while the respiratory apparatus and the digestive system were entirely free from the disease—from any symptoms indicating its presence.

General symptoms.—The disease seemed to affect more particularly one of three different groups of organs, or systems, sometimes alone, and sometimes affecting all three at the same time. Frequently the disease localized itself upon the respiratory mucous membrane, followed speedily by a catarrhal difficulty of the stomach and bowels, and accompanied by a general involvement of the nervous system. I have treated cases in which I momentarily expected a convulsion, with meningitis ; in another death seemed imminent from the exhaustion following severe vomiting, and fifteen or twenty bowel movements daily ; while the next would seem in a dangerous and critical condition from its rapid breathing and obstructed circulation. Almost a constant observation that occurred in my practice, was either a redness of the face, with

a tired or sleepy expression occurring at the same time, with redness of the conjunctiva, and of all the mucous membranes in sight, or an appearance of pallor and great depression with a feeling of weariness. This last symptom was particularly marked in many young and feeble children.

The severe chill which is noticed as a first symptom in adults, does not, so far as my experience goes, become manifested in the case of children. In almost all epidemics—and notably in the one I am describing—earache is mentioned as a prominent and frequent symptom. Indeed, associated with a catarrh of the upper passages, it is given as a prominent diagnostic feature.

Temperature:—Some authors make a distinction between a mild and severe form of the disease based upon the temperature, the milder forms having a temperature not to exceed 102 degrees F. in the afternoon, while the severer forms reach 104 degrees, or higher, at the same time of the day. In a few cases fever seemed to manifest itself only at one time during the day, and if but a single professional visit was made to the patient, the physician was likely to escape the temperature markings of the case. Many cases reached a temperature of 105 degrees in our epidemics, and one physician records cases showing 105.4 degrees, while another speaks of a temperature of 106 degrees. High fever, in my judgment, was not generally a noticeable feature in our cases—that is, a high temperature would not persist to such a degree, and for a long enough period to make it a possible cause of death. A high temperature, or at least a severe temperature, persisted for a long time during the convalescence of some cases, making us fearful lest there might be developing some systemic affection.

Particularly during the epidemic of 1891 * did there come under my notice some rare instances of subnormal temperature, and extremely wide range of temperature—as great a range, in transient disease, and not ante-mortem,

* Epidemic of 1891–92.

as I can recollect of ever observing. I have the notes of one case in which, the second day after a temperature of 104-5 degrees was chronicled, the thermometer, in the same situation, would only register 96 degrees; and two days afterwards 93 *was the highest axillary temperature to be obtained.* In this case I have a showing of six days, during which the temperature at no time reached the normal point, varying from 93 to 98 degrees F. Furthermore, the system appeared not greatly disturbed, the pulse being 90 to 100, and the child at times normally playful and bright.

P. Malling Hansen, of Copenhagen (*Archiv. für Kinderheilkunde*, 1891, p. 84.) gives some very interesting figures indicating somewhat the constitutional effect of a full play of the resisting power of the body, or the influence of an undeveloped invasion of the specific cause of the disease. Studies in this direction will be valuable if they can be followed up.

Hansen says: "In the asylum for deaf and dumb at Copenhagen, all scholars are weighed daily. The figures do not give the weight of the individual child, but of groups. In 1889 the curves were usual. From Nov. 22 to Dec. 21st, 1890, the usual increase in bodily weight ceased, in girls entirely; in boys there was only 200 gr. instead of the usual 700 gr. per month. In feeding and all local conditions there was no change during this time. *Influenza was prevailing in Copenhagen during this period.*" The author's conclusions were that this peculiar standstill in the normal increase of weight represented the conflict between the influenza poison and the subjects, and that all had the disease, *although none of them showed other signs of its presence.*

Symptoms referable to the respiratory system:—A catarrh of the respiratory apparatus takes place with as great frequency as any other one symptom, commencing with the nose and upper respiratory passages and rapidly spreading so that within an extremely short time the entire air tract is involved. We have sneezing and evidence of involvement of the Schneiderian membrane, with a

cough, particularly of a tracheal character, which is soon followed by an apparent localization of the infection in one or both of the lungs, threatening a pneumonia. I have frequently noticed, however, that the following day a general bronchitis is found to be present, and the localization has disappeared. A general catarrhal bronchitis seems to be as frequent a complicating disease as was developed in the course of the late epidemic. The prevalence of a general catarrh of the bronchial tubes, which does not develop into a pneumonia, is an observation which should be remembered. We have, too, a severe and perplexing cough without any physical signs. Respiration is sometimes very slow, and a few cases have occurred in which the respiratory efforts have absolutely stopped for a few seconds, these peculiar paroxysms coming on several times during the day, and the life of the child has only been preserved during such times by the employment of artificial respiration.

Symptoms referable to the circulatory system :—Depression of the heart, and failure in its nerve supply, seem always out of proportion to all other symptoms. There is usually from the first a rapid and weak heart, and fainting takes place in many cases. The pulse is rapid and weak, and oftentimes intermittent. In some patients the temperature and pulse seem fair, but a general muscular weakness, with a tendency to faint is prominently noticed. One child had an organic heart disease after the influenza which had never been recognized previously. Cyanosis is sometimes present, and the weak heart is so manifest that death seems imminent.

Symptoms referable to the digestive apparatus :—Loss of appetite, with persistent vomiting, was noticed in many cases. Diarrhœa was a troublesome symptom in a large number of children, constipation present in a few instances; tongue flabby and coated and showing teeth indentations, indicating a state of malnutrition.

Symptoms referable to the nervous system :—Great irritability and fretfulness was almost always present, and if these little children could speak they would tell us of

headaches and hyperæsthesia of the scalp, and of joint and muscular pains. I have no doubt concerning these symptoms, for in many cases, although the greatest care was observed, they seemed to suffer whenever moved. Convulsions took place in a small per cent. indeed there was some impairment of the nervous system in about all the cases which I have observed. There was an indifference —a hebetude—noticed in a large number of cases. Congestion of the brain, with drowsiness, appeared with many, and in one case under my observation a child never closed its eyes for four days and nights. This child was not unconscious, it was not comatose, but indifferent—wanted to be left alone. Chorea has followed a number of times.

Next to the catarrhal symptoms, one of the most peculiar manifestations noticed was sleeplessness at night, and more or less excitement during the day. Little patients were remarkable in being obstinate, protesting against the slightest touch, and refusing the slightest examination on the part of the physician. This peculiarity is regarded as of diagnostic importance in differentiating from typhoid fever; in typhoid the child is gentle, indifferent. The extreme irritability of the nervous system makes the attendant fearful of meningitis. Acute mania has actually followed; in one case it persisted for over a week, and a second child, a girl fourteen years of age, was thus affected.

Complications.—Glandular enlargements take place with frequency, and tenderness of glands is of rather common occurrence. Inflammation of the parotid gland, simulating mumps, has developed, and in one case the glands of the throat in the vicinity of the bronchial tubes became swollen and tender. Abscess of the antrum has also taken place, and the connective tissue of the neck has become sensitively affected. Tuberculosis and tubercular meningitis followed in a few cases. Indeed, Unger makes the statement that these tubercular complications are particularly to be feared.

Hyperpyrexia, without evidences of local involvement sufficient to account therefor, and of a recurring nature, I have observed, particularly in one case wherein the body

heat repeatedly went to 105 degrees F., and in which the illness continued over a period of two or three months.

Dr. Boerne Bettman, of this city, kindly informs me that he has most frequently observed a simple conjunctivitis, similar to that seen in scarlatina and measles, and that he considered such condition secondary to the rhinitis accompanying the influenza. This observer also found many cases of catarrhal inflammation of the middle ear, resulting, in a few instances, in perforation of the membrana tympani, and profuse discharge. In most cases, however, there was not more than a condition of congestion of the drum membrane, which readily yielded to treatment. In neglected cases of both ocular and aural disturbances very much more serious results subsequently occurred, more properly classified as sequelæ than complications.

Diseases of the skin have also been noticed, such as, more particularly, erythema, herpes, urticaria.

Among the more general diseases noticed, have been rheumatism, nephritis and periostitis. Children having a tendency to rickets have been found to develop the disease with greater frequency and readiness after having an attack of influenza.

Diagnosis.—From simple catarrh *la grippe* will be recognized from the fact that there is greater prostration, that the disease continues for a longer period of time, and we appreciate that we are in the presence of an epidemic. The temperature is higher, and there is a tendency for the catarrh, which is at first local, to rapidly spread to other parts of the body. A severe and irritating cough, which resists all methods of treatment, if it occurs during an epidemic of influenza, should be classed as *la grippe.* A mild catarrh with rather severe neuralgia, with pain in the head and limbs, should also be included, and the same may be said concerning an irritable stomach with diarrhœa, particularly if we are sensible of an epidemic prevalence.

The disease is to be distinguished from typhoid fever by the fact that in influenza there are no rose spots, swel-

ling of the spleen is unusual, and the catarrhal difficulties, particularly of the respiratory tract, predominate above all other symptoms. If diarrhœa exists, as it does in many cases, it will be noticed that a cough and a catarrhal state of the air passages has preceded its development. The fever in influenza is irregular, while in typhoid it has a course nearly diagnostic of the affection. Typhoid fever is rare in children under two years of age; influenza is rather frequent within these boundaries. From pneumonia, we differentiate by the absence of the usual physical signs, although at the onset of *la grippe* there may be every evidence of a pneumonia, yet in a few hours the localization has become diffused, and a genuine (and more or less general) bronchitis is the result. The presence of meningitis can usually be detected by careful observations of the eye, the rigidity of the muscles, and other symptoms nearly always present—although in my judgment the differentiation of meningitis from severe forms of *la grippe* is attended with trouble, and a diagnosis must fre quently be held in reserve for some time. The different manifestations of tubercular disease which follow in influenza, will be made out by carefully watching the temperature and respirations, observing the influence on the general nutrition, and by the development of tubercles on the choroid.

Prognosis and Mortality.—The mortality is different in different epidemics, and the character of the epidemic must be considered the same as we do in other infectious and contagious diseases of children. Death takes place from almost every complication; in the main, however, it is exhaustion and bad nutrition that we have most to fight. It is claimed by some that apoplexy occasionally causes death.

The length of time consumed in the convalescence from this disease is rather wonderful. The pains and general weakness do not disappear for weeks, and as I remark in another place, a fever may continue for months, and without doubt many of the sequelæ will remain for years, if not throughout the lifetime of the individual.

One significant fact which presented itself to me, and to which my attention has been later attracted in an examination of the literature bearing upon this subject, is that children do not suffer from this disease until late in the epidemic.*

In the city of Chicago, closely following this terrible epidemic which prevailed alike among children and adults, has followed a most severe epidemic of typhoid fever. Just what its relation to the previous occurrence of influenza may be, I cannot at this time state. Children were affected with *la grippe* and their general nutrition greatly reduced, then followed a large number sick with the typhoid fever; and now, whether there was a direct relationship between the two diseases, or whether the systems were weakened by the first affection so as to make it more easy for the typhoid to gain entrance, I reiterate, cannot now be unquestionably stated. Dr. Foster writes me his belief that the poison of influenza so weakened mucous surfaces that the germs of typhoid had an easy and favorable nidus. It is hoped that future observations will enlighten us on this subject.

Somewhat in this line of thought, but not directly allied to my subject, is the question of premature births and abortions. An examination of the literature finds the statement that pregnant women—under the influence of epidemic influenza—are more liable to premature deliveries. It was noticed by some of my correspondents that rather an unusual number of cases aborted, and that premature labors were somewhat more frequent. I am certain that the prevalence of *la grippe* in the persons of our puerperal patients produced fever which continued for many days, and that the catarrhal symptoms, with the irritating and relentless course, made their convalescence very slow and tedious.

Treatment.—If the disease is of bacterial origin then great care must be taken with regard to its prophylaxis. All handkerchiefs and cloths used by the patients must be

* This fact was not noticed in the epidemic of 1891–92.

immersed in some antiseptic liquid, and all cuspidores and articles of furniture which come in contact with the germs which may produce this disease, should be carefully disinfected.

As far as my observation has extended, there is no specific treatment for this disease, but I should say that a *generous diet*, with some stimulation and a conservation of all the strength of the patient, should be observed from the first—and just as carefully as we give attention to these conditions in typhoid fever. For the general pain which pervades the entire system, nothing has given as good results, in the cases falling under my care, as phenacetine, either alone or in combination with salicylate of soda, and, in certain cases where the fever is high, and where the strength will allow it, moderate doses of antipyrin.

For the extreme fatigue and depression which children evince, milk-punch, with some of the effervescing waters, together with small doses of quinine and a generous diet, seem to give the best results. Whenever quinine was used it was administered by inunction or by the rectum.

The plan of sending city children into the country has nothing to commend itself to me, for the disease raged with quite as much severity in the outlying districts as in our city.

If the catarrhal difficulties impede children from nursing, then their nutrition must be kept up by artificial means.

By way of conclusion, I feel that I cannot too strongly emphasize the necessity of conserving the vital energy of these little sufferers when laboring under the very markedly depressing influences pertaining to this disease; I am thoroughly convinced of this point, and the value derived from carrying out this principle.

I would advise the early use of equal parts of whipped egg-albumen and sterilized water, with a little brandy and sugar, varying the amount of the alcoholic according to the condition, age, and individual susceptibilities of the patient. This should be exhibited often and with regularity,

and in such quantity as to be readily assimilated. As the child begins to pass out from the more painful and acute manifestations of the disease, there may be given, in addition to the generous diet, the compound syrup of hypophosphites, together with pepsin cordial and a little nux vomica. This more particularly in the way of a tonic, or "bracing agent." Where the lungs are particularly involved—and they often become very rapidly involved—the more pronounced diffusible stimulants have an important place. Here the carbonate of ammonia is of service, also, aromatic spirits of ammonia, alcohol, camphor and musk, and either, or all, are well fortified by the conjoint use of small doses of digitalis and nux vomica. In these lung complications the same general tonics and reconstructives may be used during the period of convalescence. Malt and hypophosphites, also, malt and cod-liver oil are first-class agents for this purpose. In the gastro-intestinal form even a greater degree of attention should be directed to the diet. Everything passing into the stomach should be simple and very easy of digestion —pre-digested so far as can be. It becomes often necessary to avoid all milk, using, instead, rice-water with a little cream, and a small quantity of wine of reliable vintage. Where there is great, or even moderate, gastro-intestinal fermentation large doses of bismuth, combined with a moderate amount of salol, has given the best results in my hands.

I have found some cases—the exact proportion to the whole number I have not determined as yet—where there was as profound and sudden collapse as appears in severe cases of cholera infantum ; where, in a few hours, the patient would be placed upon the very border-lines of dissolution, demanding the prompt use of external heat, and the active diffusible stimulants.

Since this paper was read, and more particularly while it has been in the hands of the printers (February, 1892), several important communications have been received. It is impossible to incorporate these recent investigations which, though they would not change my general obser-

vations, would add much to our knowledge of this disease. The study has been along the line to discover a cause for these terrible visitations which, possibly, has been successful, but I must be content to refer the reader who desires additional literature on the subject to the *British Medical Journal*, January 16th, 23d and 30th, and to the *Medical Record* of January 30, 1892.

A CASE OF ULCERATIVE CATARRHAL DYSENTERY.

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THE discovery of amœba in dysenteric discharges, associated with a definite clinical history and characteristic lesions in the intestine and other organs of the body, marks an epoch in the history, of this important class of diseases. So long as we were dependent upon the clinical history and anatomical lesions alone to distinguish between diseases so closely resembling each other, as the different forms of dysentery, it was impossible to have clear views concerning them, but when we can add to this means of differentiation a special etiology for one form of the disease, much of the confusion is removed.

Amœba were first observed in intestinal discharges by Lambl, in 1860, in the stools of a child affected with enteritis, but no importance was attached to these organisms until 1872, when Lösch found them in a case of dysentery. Lösch thought amœba either caused the disease or proved a source of irritation, and prevented the ulcers from healing.

Since attention has been drawn to amœba by the views of Lösch, they have been found in dysenteric discharges by numerous observers in different parts of the world, and

the recent work on this subject by Councilman[2] and Lafleur establishes almost beyond doubt, that amœba cause the disease previously known as tropical dysentery, and that this disease is by no means confined to tropical countries, but is also not uncommon in this country and in Europe.

With the other forms of dysentery now recognized, viz., catarrhal and diphtheritic, we have not been so fortunate. Nothing is known of a specific exciting cause.

Amœba have not been found in catarrhal and diphtheritic dysentery having relation to the anatomical lesions; and from the nature of the lesions in these forms of dysentery, it is probable we have to look for an origin from bacteria rather than amœba. It has been supposed for many years that bacteria play an important part in dysentery. In 1869 Hallier observed a number of micrococci in dysenteric discharges, which he claimed to have differentiated by culture methods from the micrococci found in the diarrhœal and healthy fæces. But the methods in use, at that time, for separating bacteria were so imperfect, that no reliance could be placed in his assertion.

Since the introduction of trustworthy methods for separating bacteria, considerable attention has been paid to bacteria in the diarrhœal fæces, but very little attention has been given to those in dysenteric discharges.

There has not, to my knowledge, been a case of dysentery in which cultures have been made of bacteria in the intestine and other organs of the body after death, and only a few cases in which bacteria have been isolated from dysenteric stools.

Conclusions cannot be drawn from a single case, and such is not intended in this report. With the advantage of an autopsy in thirty minutes after death and cultures made from the abdominal organs in one to three hours after death, the record of a single case carefully worked out under such favorable circumstances may be of importance for future comparison.

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[2]Johns Hopkins Hospital Reports, 1891.

Henry L., twenty months old, was admitted to the Thomas Wilson Sanitarium, July 20, 1891. One week before this he was taken sick with vomiting and purging. The stools were not frequent for the first few days and were composed chiefly of yellow fluid. On the fifth day the stools became more frequent, and contained mucus and blood, and were accompanied with pain and straining.

He was the second child in the family. The other child died of dysentery when two years old. Parents healthy. This child had previously been healthy with exception of slight colds, and for three weeks prior to the present sickness he had at times a tendency to loose bowels, but never more than two or three loose stools during the day, and this only for a few days at a time.

He had been nursed at the breast, and in addition given food from the table, including cabbage, tomatoes and other vegetables. The day before this sickness commenced, he ate some blackberries, which were discharged from the bowels almost in the same condition they were when swallowed.

Condition of patient when admitted to the Sanitarium.—Body greatly reduced in flesh; ribs beaded; chest sounds normal; pulse rapid and feeble; temperature 101°; appetite poor; child restless and fretful; stools green, and contained lumps of mucus and blood; fifteen stools during the day.

He was under observation at the Sanitarium eleven days. His stools were not very frequent after the first day. He had eight stools on the 21st, twelve on the 22d, and after this from two to eight stools daily. The stools were often small, consisting of lumps of mucus and blood, and showing under the microscope chiefly blood corpuscles and pus cells, with comparatively small number of bacteria. The pain at stool was not very marked, and was manifested by restlessness or whining, and not by loud cries. The child was sometimes very restless and cried in sleep as if in pain; generally he laid in a listless and stupid condition. He had no appetite, and often had difficulty in swallowing. From the 20th to 26th of July his temperature ranged from 99° to 101.8°. After this time there was a gradual and almost steady rise in the temperature until the 30th, when it reached 104°.

Death July 31, at 2 A. M. Autopsy at 2.30 A. M.—Body emaciated. Heart and lungs appeared normal except that both were very pale.

Stomach.—Contained about one ounce of dirty, milk-like fluid, which showed under the microscope immense number of bacteria, some of which were micrococci, chain bacilli and forms like bacterium lactis ærogenes. There were also a few sarcina.

Duodenum.—Nearly empty and had an invagination, one and one-half inches long, a few inches below the pylorus. Contents of the duodenum were brown and pulpy, which, examined under the microscope, were found to contain cylindrical epithelial cells and few bacteria. The most numerous form of bacteria resembled bacterium lactis ærogenes. There were few of the micrococci and chain bacilli seen in the stomach, and few forms resembling bacterium coli commune.

Jejunum.—Slightly distended with gas, and had a small amount of greenish pulpy substance in places, which was found on microscopical examination to contain epithelial and small round cells, and many bacteria. Forms like bacterium lactis ærogenes predominated. There were also a few small rods, micrococci, and some like bacterium coli commune.

Lower end of ileum.—Contained bloody, purulent fluid, which showed under the microscope pus and epithelial cells, blood corpuscles and enormous quantity of bacteria of many different forms.

Colon.—The upper end of colon contained large quantity of white purulent fluid, the lower end bloody purulent fluid. Microscopical examination; pus cells, blood corpuscles, and large number of bacteria, which were mostly forms like bacterium coli commune, shorter and thicker rods, and some smaller rods.

The following report is from Prof. Welch's examination of the abdominal organs:

The entire length of the large intestine, from cæcum to anus, is extensively ulcerated. The prevailing ulceration is superficial, involving, apparently, not more than mucous membrane. The ulcerated part has a smooth, grayish appearance, with irregular margins, the remaining mucous membrane standing as islands, and larger or smaller areas, elevated, brownish-red, soft, velvety, highly congested, on this grayish ulcerated surface. At first glance it is not altogether easy to say whether there is any ulceration (except the small ones noted below), but frozen sections show the ulceration to be so extensive in places, for instance, just above the anus, that only here and there a small islet of mucous membrane remains.

There is no undermining of mucous membrane. The process involves the whole length of large intestine, with about equal intensity, perhaps rather more extensive, below. There are in places small, round, deeper ulcers, generally not more than 2-3 mm. in diameter, easily recognized as ulcers, extending into the sub-mucosa.

The Peyer's patches in end of ileum are somewhat swollen and surface is irregular, the last one may be superficially ulcerated, but not positively to be recognized as ulcerated.

Liver, firm, pale whitish-yellow. Frozen sections show intense fatty metamorphosis, involving the whole liver, the liver cells being filled with medium-sized and small oil drops. The process is in many lobules most intense around centre of lobule and in peripheral zone, leaving intervening middle zone less affected.

Kidney rather pale to naked eye. Some opaque whitish dots and streaks in cortex and pyramids visible with naked eye on frozen sections, which, microscopically, show considerable fatty degeneration and cloudy swelling of renal epithelium. That of convoluted tubules much affected, likewise tubes in pyramids. Some fine fatty granules in glomeruli. Some tubes, especially in cortex, contain irregular, homogeneous refractive material.

Great number of bacteria seen in surface of frozen sections of intestine, which show the surface of the ulcerated part richly infiltrated with small, round cells, with only moderate deep infiltration of sub-mucosa at this place, and apparently none where mucosa is intact. There is neither microscopically nor to the naked eye any trace of diphtheritic deposit.

The process appears to be a suppuration or infiltration of mucosa with leucocytes and breaking down of the superficially infiltrated parts.

Examination of the hardened sections by Dr. Councilman :

Lower end of ileum passing through the last Peyer's patch. Over considerable portion of section there is loss of substance of upper layer of mucous membrane. In very few places this involves the entire thickness of mucosa, extending to muscularis mucosa. The interglandular tissue contains an excess of lymph cells with a few leucocytes. The glands are generally filled with mucus, and in some a few leucocytes are found. Some small cell infiltration of sub-mucosa. Muscularis mucosa unchanged.

Colon.—Changes much more extensive and characteristic, almost confined to mucosa. In places the glands are swollen, filled with mucus and pus cells, and show extensive desquamation. The interglandular tissue also contains a number of leucocytes. There is extensive loss of substance, sometimes involving only the upper layer of mucous membrane, sometimes the whole thickness of this down to muscularis mucosa; for a considerable distance this may be laid bare. In some places there are enormous dilatation of blood vessels of mucous membrane. In only a few instances has the process extended into the sub-mucosa, when it has, there is also here a shallow loss of substance. In the sub-mucosa there are in places slight thickening, swelling of connective tissue cells and granulation cell infiltration.

The lymph follicles are not involved in the process at all.

The whole process appears to be one commencing on the surface and slowly extending downward.

Bacteria isolated from contents of stomach and intestine, and other organs:

Roll-tube-cultures were made in one to three hours after death, from the stomach, intestine, mesenteric glands, liver, gall-bladder, kidney, spleen, and blood from the heart.

Stomach.—1st tube cloudy from number of colonies; 2d tube thick, but colonies isolated, chiefly colonies of bacterium lactis ærogenes; many colonies of a slow liquefier, none of bacterium coli commune.

Duodenum.—1st tube cloudy; 2d tube thick. Almost a pure culture of bacterium lactis ærogenes.

Jejunum.—1st tube cloudy. Chiefly bacterium lactis ærogenes; many of bacterium coli commune.

Upper ileum.—Colonies numerous, many of bacterium lactis ærogenes and bacterium coli commune. Few colonies of a new species of bacteria.

Lower ileum.—1st tube cloudy. The new bacillus predominated, bacterium coli commune and bacterium lactis ærogenes numerous and nearly equal in number.

Colon.—Tubes very thick; bacterium coli commune predominated, many of the new bacillus; many colonies of a bacterium, closely resembling bacterium coli commune; few of bacterium lactis ærogenes.

Mesenteric glands.—Gland opposite duodenum, one colony of bacterium lactis ærogenes; gland opposite upper end of colon, about one hundred colonies; twelve of these

were bacterium coli commune, the other colonies were the new bacillus.

Blood from the heart contained a pure culture of the new bacillus, but the number was not estimated, owing to liquefaction of the tube from atmospheric heat. No growth on tubes from the gall-bladder and spleen. Tubes from liver and kidney were rejected.

General considerations :

1. Comparison of the bacterial vegetation in this case with that of the healthy and diarrhœal intestine of sucklings.

Healthy intestine examined by Escherich.—Two species of bacteria constantly present, viz., bacterium lactis ærogenes and bacterium coli commune. These two species have a constant relation to certain parts of the intestine: bacterium lactis ærogenes being most abundant in upper part of intestine, and often a pure culture in the duodenum. It diminishes in number in passing down the intestine, and but few are found at lower end of ileum, and still smaller number in colon. Bacterium coli commune greatly predominates over all forms of bacteria in the colon, and often a pure culture there. It is often not found at all in the duodenum; is sparse in jejunum, but becomes more abundant in the upper end of ileum, and at lower end of ileum greatly exceeds bacterium lactis ærogenes in numbers.

Diarrhœal intestine.—In five cases which I have examined the relation of the two above-mentioned species of bacteria in the intestine differs from that found by Escherich for the healthy intestine. Bacterium coli commune was almost as abundant in the duodenum and other parts of small intestine as bacterium lactis ærogenes, and the latter was more abundant in the colon than appears in the healthy condition.

In this case of dysentery, bacterium lactis ærogenes and bacterium coli commune correspond in their distribution in the intestine more with the healthy than the diarrhœal intestine.

Bacterium coli commune and bacterium lactis ærogenes are pathogenic in lower animals. Whether they become in-

jurious to children, and the conditions under which this may happen is not known. The determination of their true pathogenic properties is one of the most interesting problems of bacteriology, and is at present attracting considerable attention. There are reasons for considering them more capable of power for harm than their constant presence in the healthy intestine would lead us to suppose.

2. The presence in this case of a new species of bacteria in large quantity in the diseased part of the intestine and its absence in the healthy portion. This species of bacteria differs from all the species which I have isolated from the contents of the rectum of eighty-five children affected with diarrhœa, and does not correspond to the description of the bacteria isolated by others from the diarrhœal fæces. In morphology it resembles bacterium lactis ærogenes, but is slightly smaller, and forms nearly round, like cocci, are more often seen. The gelatine colony-growth also resembles bacterium lactis ærogenes. The spread, surface colonies have the same loose brownish-yellow appearance, but in this species there is a knob-like central point, which gives a button-like appearance. The deep colonies differ more from those of the bacterium lactis ærogenes in having a beautiful concentric arrangement, but this is not always present. The milk-litmus reaction is characteristic. The litmus gradually fades, and in three to five days in the thermostat the milk is coagulated into a soft clot. The change of litmus to red requires a longer time, and only takes place on top of the clot. This species of bacteria produces fermentation in milk.

3. The presence of a species of bacillus, not previously recognized, in considerable quantity in the colon. This species resembles bacterium coli commune in many respects, but differs from it in not coagulating milk or reducing litmus.

4. The large number of bacteria in the stomach.

5. The large number of bacteria found in the mucous membrane of hardened sections of colon. Most of the forms seen resembled bacterium coli commune, many resembled

the new bacillus, but these were nowhere so abundant as bacterium coli commune. The latter are found in great abundance in some places, and extended through the whole depth of the mucosa. In some places the new bacillus was found in considerable clusters.

6. The distribution of the bacteria in the mesenteric glands. In the duodenum bacterium lactis ærogenes was almost exclusively found, and in the gland opposite the duodenum the only colony growing from the culture was bacterium lactis ærogenes. In the colon where the new bacillus and bacterium coli commune were in greatest abundance, the glands of the colon contained considerable numbers of these two species, and no other forms of bacteria.

SCARLATINAL NEPHRITIS.

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NEPHRITIS is a very important and common complication and sequel of scarlet fever. It usually begins in the declining period, or during convalescence, in mild as well as in severe cases. It is sometimes slight, producing but little aggravation of symptoms, but in other instances it changes the prognosis, causing death in cases which seemed to be favorable prior to its occurrence. The presence of albumen in the urine, which not in all instances, but ordinarily indicates the occurrence of nephritis, is more common in some epidemics of scarlatina than in others. In the *British Medical Journal*, 1885, Mr. Thomson states that albumen occurred in sixty per cent. of 180 cases. Microscopic examination also revealed the presence of casts and blood cells in most of these cases. Heidulhane found albuminuria in eighty per cent. of the scarlatinous cases examined by him. On the other hand, Charles West, of London, discovered albuminuria in only four per cent. of his patients in one epidemic. It commenced most frequently at the end of the first, or in the second week. Atkinson, in a paper published in the

International Journal of Medical Science, July, 1886, says in some epidemics albuminuria is present in seventy per cent. of the cases, and in other epidemics, in not more than five per cent. He makes the remarkable statement that Jaccoud has not seen a case of scarlatinal nephritis in fifteen years, and that he (Jaccoud) attributes the absence of this complication largely to the use of a milk diet. In New York City, so far as my observations extend, in every scarlatinous epidemic a daily examination of the urine in each case at the close of the first week and subsequently, reveals the presence of albuminuria in more or fewer cases, the proportion of those having albuminuria varying in different epidemics. The London *Lancet*, Nov. 26, 1887, states that the statistics of 3,000 cases of scarlet fever admitted into the London institutions, showed a death-rate of seven per cent. Albuminuria in an appreciable quantity, and for a considerable time, occurred in fifteen to twenty per cent. of these cases. Eighty-three scarlet fever patients died in two of the hospitals, and some form of kidney disease was responsible for this result in twelve per cent. of the deaths.

Anatomical Characters.—Dr. William H. Welch, the distinguished professor of pathology in Johns Hopkins University, wrote as follows in a note bearing the date September 16, 1891: "In regard to the renal complications of scarlatina, we have probably to distinguish the moderate transitory albuminuria accompanying the early stages, and the height of the disease from the genuine scarlatinal, or post-scarlatinal nephritis. The former is probably associated only with such parenchymatous changes as accompany many acute infectious fevers, and is not a genuine nephritis. The latter is, in most cases, a glomerulo-nephritis, with a varying amount of interstitial change in the form of small-celled infiltration. Whether the genuine scarlatinal glomerulo-nephritis is caused by the specific virus of scarlatina, or by some complicating secondary organism is not settled."

Prof. Francis Delafield, of New York, of the College of Physicians and Surgeons, has made many microscopic ex-

aminations of inflamed kidneys, and he writes as follows of acute exudative nephritis: "This is frequently a primary nephritis, either occurring after exposure to cold, or without discoverable cause. It complicates scarlatina, measles, diphtheria, typhoid fever, acute general tuberculosis, pneumonia, acute endocarditis, acute peritonitis, dysentery, erysipelas, diabetes, and many other of the infectious diseases and severe inflammations." He states that it has the characters of an exudative inflammation. The liquor sanguinis and the red- and white-blood cells escape from the renal vessels into the tubules. "Swelling or necrosis of the renal epithelium, and changes in the glomeruli" may also occur. In severe cases resulting fatally, "we find the kidneys large and smooth, the cortex thick and white, or white, mottled with red, or the entire kidney intensely congested. If the stroma is infiltrated with serum the kidney is succulent and wet; if the number of pus cells is very great there will be little whitish foci in the cortex." The tubal epithelium is sometimes swollen and opaque. Hyaline cylinders, identical with the casts, are found in the convoluted tubes, and more abundantly in the straight tubes, along with irregular masses formed from the exuded blood plasma. In the tubes are also red and white-blood cells. The glomeruli exhibit important changes. They become larger and more opaque, "due to the swelling and growth of the cells on and in the capillaries," for the glomerular capillaries in their normal state are covered on their outside by nucleated cells, and flat cells line their inner surfaces in places, not continuously. On account of these cellular changes, the individual capillaries in the glomerulus become indistinct, but "the main divisions of the tufts are visible. . . . In very severe cases the growth of the cells on the tufts is so considerable that they form large masses of cells between the glomerulus and its capsule. The walls of the arteries in the kidneys may be thickened by a swelling of their muscular coats." Acute exudative nephritis may be fatal, even rapidly, but in general, those who recover, recover completely. The structural changes enumerated above

are more marked in the cortex than in other parts of the kidney.

Acute Diffuse Nephritis.—Dr. Delafield says in regard to this disease: "It is one of the forms of scarlatinous nephritis. . . . The kidneys are large, at first smooth; later sometimes a little roughened; the cortical portion is thick, white, or mottled with yellow or red, or congested; the pyramids are red. In these kidneys we find the same lesions as have been described as belonging to exudative nephritis, but with two additional changes, changes which are found in the earliest stages of the inflammation, and which give the characteristic stamp to the lesion. First, a growth of connective tissue in the stroma; second, a growth of the capsule cells of the glomeruli. These changes do not involve the whole of the kidney, but symmetrical strips or wedges in the cortex, which follow the line of the arteries. These wedges are small or large, few or numerous, regular or irregular, in the different kidneys, but in every wedge we find the same general characters; one or more arteries, of which the walls are thickened; glomeruli belonging to these arteries, with a large growth of capsule; cells compressing the tufts, "a growth of new connective tissue in the stroma, around and parallel to the arteries. Between the wedges we find at first only the changes of exudative nephritis, later a diffuse growth of connective tissue. If the nephritis is of acute type, and longer duration, the tissue is denser, and has more basement substance. Where the growth of the new tissue is abundant, the tubes become small and atrophied. The exudation from the blood vessels is very considerable, so that the urine contains large quantities of albumen, many casts and red and white-blood cells." (Handbook of Path., Anat. and Histol., Delafield and Prudden, Wood & Co., 1889.)

The nephritis complicating and following scarlet fever is frequently more dangerous than the primary disease. A clear appreciation of its therapeutic requirements is important, since by judicious treatment many recover, who would inevitably perish by improper measures. The

family should be informed that the danger from scarlet fever does not cease with the decline of the eruption, and that the kidneys may become seriously affected when the patient is in other respects convalescent.

In considering scarlatinous nephritis, the fact already alluded to should be borne in mind, that as Prof. Welch has stated, albumen in the urine, which, easily ascertained, is regarded by many practitioners as a certain diagnostic sign of nephritis, if it occur during, or immediately after scarlet fever, sometimes, if transient, occurs from slight anatomical changes in the kidneys, not nephritis. The late Prof. Austin Flint, a very accurate clinical observer, wrote as follows: "Transient, slight albuminuria occurs in scarlet fever as in various other, without denoting renal disease (*Practice of Medicine*, page 1055). This transient and slight albuminuria, without nephritis, occurs during the height of scarlet fever, when the temperature is elevated, and the patient is seriously sick in other respects, than as regards the urinary apparatus. Owen Fowler, in his remarks on 2,000 cases of scarlet fever occurring in four years in the London Fever Hospital, states that albuminuria was sometimes due to the high temperature (*British Medical Journal*, Nov. 14, 1885). The nephritis which results from glomerulo-nephritis, and indicates its presence, occurs for the most part when scarlatina is abating or has abated, and it continues as a sequel. Unlike the transient albuminuria alluded to by Profs. Welch and Flint, it is attended by the presence of more or fewer blood cells and casts in the urine.

The cause of scarlatinal nephritis is an interesting subject for investigation. Prof. Flint wrote, "It is an important question, whether this sequel proceeds exclusively or chiefly from an agency pertaining intrinsically to scarlatina, or whether it depends on extrinsic causes, such as the action of cold." Mr. Thomson, in a paper published in the *British Medical Journal*, Nov. 14, 1885, quotes the opinion of Mr. Dickinson, that only a small proportion of the cases of scarlatinal nephritis are due to taking cold.

The theory which is accepted, relating to the ætiology of a disease, influences its treatment, and the theory that scarlatinal nephritis is not caused, or is rarely caused by "taking cold," and that it is commonly caused by microbic agency, is likely to lead to the abandonment of the precautionary measures against "taking cold," long recommended and employed by the profession. The opinions of the distinguished physicians whose names we have mentioned, favorable to the microbic theory, must have their influence. Moreover, we have stated in a paper already published, that bacteriologists have discovered microbes, the streptococci predominating at the seat of inflammation in the otitis, arthritis, pleuritis, pneumonia, pericarditis and endocarditis, complicating scarlet fever. Besides, in thirteen of fourteen cases of albuminuria and œdema occurring in scarlatinal nephritis, it is reported that Babes found the streptococcus either alone or associated with the pneumococcus of Talamon-Fränkel in the kidneys.

On the other hand, it is well known that nearly all writers on nephritis occurring independently of scarlet fever, regard taking cold as the most important factor producing it, and from the observation extending through many years of cases of scarlatinal nephritis, I am impressed with the belief, as I have stated elsewhere, that those who are in bed, or in a warm room until the acute symptoms have abated, and are carefully housed or warmly clothed during the period of desquamation, are more likely to escape the nephritis than are those who are early and carelessly exposed to currents of air or out-door weather. According to my observations, the mildest cases of scarlet fever, in which the system is so slightly affected, that the presumption is strong that the kidneys cannot be injured by the direct action of the poison, or by microbic agency, are, nevertheless, liable to contract nephritis if too early exposed without sufficient covering, to cold air or out-door weather. Thus I recall a case of severe scarlatinal nephritis, which occurred in the favorable season of mid-summer, and was nearly fatal from

eclampsia. The parents at first said that the child had not been sick, but they finally recollected that it had had a rash two weeks previously, which they supposed was due to the summer heat, and during the continuance of which it played every day in the open air. Microbic agency in producing the nephritis in this case must have been very slight. The supposition is much more reasonable that it was caused by exposure of the child to outdoor weather during the continuance of the scarlet fever. The frequency of nephritis after mild attacks of scarlet fever is, I think, attributable to the fact that such cases are not confined to bed, or even the sick-room, and are exposed to currents of air and vicissitudes of temperature, by which transpiration from the surfaces is checked. It is evident, in the present state of our knowledge, that in order to prescribe a correct prophylactic and curative treatment in regard to scarlatinous nephritis, both theories must be accepted, that this complication and sequel is sometimes produced by microbic agency, and in other instances by taking cold.

Treatment.—The following is, in my opinion, the proper mode of treating scarlatinous nephritis. The efficacy of the following remedial measures has, I think, been abundantly demonstrated by cases occurring in family practice and in the institutions of New York. The patient should be kept in bed during the scarlet fever, however mild it may be, and in a room warm, but not uncomfortable, during the three or four weeks of desquamation. The physician should never discharge a scarlatinous patient, although his health is apparently entirely restored, without one or more examinations of his urine at his last visits. When his visits cease the nurse should be instructed to make the examinations by heat and nitric acid twice weekly during the ensuing month, and if any evidence, however slight, appear that the kidneys are involved, the physician should be notified, in order that appropriate treatment may be immediately commenced. Early and correct treatment of the nephritis is attended by much better results than delayed treatment, and many more patients are doubtless

now saved than in former times, when little attention was given to the state of the kidneys until dropsy or other prominent symptoms appeared. I have found no mother or nurse so ignorant that she could not properly employ the tests of nitric acid and heat, and if she be solicitous for the welfare of the child she will not hesitate to carry out the directions, and notify the physician if the tests employed produce the least cloudiness or turbidity of the urine.

The patient, as soon as nephritis begins, should be put to bed in a room of warm and equable temperature (72° F. to 75° F.). His diet should be liquid, consisting of milk, farinaceous food, and a moderate quantity of animal broth. He may drink liquids freely, warm or tepid, not cool, as Vichy, carbonic acid water, or plain water, to which spiritus ætheris nitrosi is added. If there be marked prostration from the primary disease, alcoholic stimulants should be allowed.

The indications of treatment are to relieve the hyperæmic kidneys by diaphoresis and purgation, and when this is effected to increase the quantity of urine, which is usually diminished, and thus eliminate the poisonous product, urea, which is the chief noxious principle generated in the system in this disease. To produce diaphoresis, the patient should be immersed in a warm bath at about the temperature of the body (98° to 100° F.), in which, if he be quiet and comfortable, he should remain from fifteen to twenty minutes, but a shorter time if restless and frightened by the water. After the bath he should be placed in a warm bed and well covered by blankets. If perspiration result, the bath has been useful, and it may be employed in grave cases two or three times daily. If perspiration do not result, it may be produced by surrounding the body by hot-dry or hot-moist air. Hot air may be produced by burning alcohol in a thin layer upon a plate under a chair, upon which the patient sits surrounded by a blanket, or he may be covered in bed and the hot air introduced under the bed clothes. In New York City a convenient apparatus is used for this

purpose, consisting of a small sheet-iron pipe enclosed in a small box of the same material. The box is in the form of a trunk, with a handle for convenience in carrying, and the lower end of the pipe, which extends nearly to the floor, contains an alcohol lamp. Hot moist air may be produced by placing bottles of hot water surrounded by towels wrung out of warm water, against the body and limbs of the patient, who is covered in bed. The steam arising from them, and enveloping the body and limbs, produces a prompt sudorific effect. A convenient apparatus for generating steam in the treatment of these and other cases requiring diaphoresis, consists of a cylinder pierced with holes for the admission of air, and containing a spirit lamp, over which is a pan or pail holding a little water. The patient, nearly denuded, is placed in a chair with the apparatus beneath, and is covered by a blanket, so that the steam surrounds the body. This gives rise to free perspiration, which continues after the patient is placed in bed. By modes of treatment like the above, which can be modified according to the exigencies or conveniences of the case, and be repeated two or more times daily, sufficient perspiration can usually be produced.

The sudorific effect of the treatment by external warmth described above, should be aided by the use of diaphoretics. Those which have usually been prescribed are the liquor ammonii acetatis, the acetate of potassium, the bitartrate of potassium, the citrate of potassium, and the spiritus ætheris nitrosi. If employed when the surface is cool, these medicines act rather as diuretics than diaphoretics. Being simple in their action and without deleterious effects, they may be given frequently and in large proportionate doses for the age. The following will be found a useful remedy, having diaphoretic, diuretic, and laxative properties :

℞. Potassii acetatis,
Potassii bicarbonatis,
Potassii citratis, aa. ʒii ;
Infus. tritici repentis, ʒviii ;
M.

Dose.—Give one teaspoonful every three or four hours to a child of five years.

But during the last few years a diaphoretic has been discovered and employed, which surpasses those previously prescribed in efficiency, and which in cases of unusual gravity, when other remedies have failed, is sometimes instrumental in saving life. I refer to pilocarpine, the active principle of jaborandi. Being soluble in water and tasteless, it is easily administered and retained, when on account of the uræmic poisoning present, the stomach is irritable and medicines that are unpleasant to the taste, are liable to be vomited. The alcoholic stimulant may be increased at the time of its use, in order to prevent any depressing effect. To a child of two years, $\frac{1}{40}$ to $\frac{1}{30}$ of a grain may be administered by the mouth every four to six hours. It may be employed hypodermically as $\frac{1}{30}$ of a grain to a child of five years. It has both a diuretic and a diaphoretic action, and it stimulates both the salivary and mucous secretions. According to one observer, an adult when fully under the influence of pilocarpine secretes from one to two pints of saliva in two hours, and Leyden reports a case of diphtheritic nephritis in which the quantity of urine rose from half a pint to five pints daily. But its most prompt and certain action is on the sweat glands. Hirschfelder speaks of its beneficial action in relieving various forms of dropsy, and adds: "In one morbid condition of the kidney, however, jaborandi is the remedy *par excellence*, and that is the acute parenchymatous nephritis which frequently follows scarlet fever. . . . . This disease heals spontaneously if the danger which threatens life from the reduction of urine, and from the effusion of fluid into the cavities of the body be averted. In this disease jaborandi works wonders." The following cases in which the beneficial action of this agent was apparent, occurred in my practice:

CASE I.—G., male; æt. five years and six months; sickened with scarlet fever, June 2, 1882. The case progressed favorably, and during the convalescence my attendance ceased. On June 24th, my attention was again called to the child, when the urine was found to be scanty and very albuminous. The common treatment was em-

ployed, to wit, warm baths, derivatives over the kidneys, laxative doses of jalap and the potassium bitartrate. The urine, however, remained scanty—f. ℥ij in twenty-four hours—and on June 28th, severe convulsions occurred, which were controlled by doses of bromide of potassium and five grain clysters of hydrate of chloral. The muriate of pilocarpine was now given in doses of $\frac{1}{32}$ of a grain every three hours in cold water. This was not vomited, and it must have been given in larger doses than that directed, for on July 1st the bottle containing one grain was empty. The mother stated that the child had taken only two doses, or $\frac{1}{16}$ of a grain of the pilocarpine, when both the diuretic and diaphoretic effects were apparent. She also stated that the quantity of urine was larger when the pilocarpine was administered every third hour than when given at a longer interval. A flaxseed poultice dusted with mustard was also applied over the kidneys. Occasional convulsive attacks continued to occur, which were readily controlled by enemata of hydrate of chloral. On June 30th, the symptoms were all better; no more attacks of eclampsia had occurred and the urine was more abundant and less albuminous. The mother remarked that the new medicine had settled the stomach and increased the urine. The record for July 4th, states: "Continues to improve; takes the muriate of pilocarpine, gr. $\frac{1}{32}$ every six hours, and has not vomited since he began to take it; pulse, 106; temperature, 99°; is playful; passes urine freely, and takes nearly three quarts of milk daily, with some farinaceous food. July 6th, is fully convalescent; pulse, 92; temperature, 99°; perspires much; urination normal in quantity and character."

CASE II.—Mary S.; æt. five years; was exposed to her brother who had scarlet fever, and about the same time she had sore throat without any efflorescence. Nearly two weeks subsequently (Dec. 22, 1882), she had the symptoms of severe nephritis; her urine was reduced to f. ℥iv. in twenty-four hours, and was highly albuminous. A powder of calomel, gr. iij., and of resina podophylli, gr. $\frac{1}{6}$, was prescribed, and it produced one stool. One-twentieth of a grain of muriate of pilocarpine administered was vomited, but another dose was taken at 10 P.M., and the mother says that the patient "sweat fearfully" in the night. Three or four doses were administered daily during the following week, and when not vomited they usually produced perspiration, lasting from one to one-and-

a-half hours. The record for December 30th, is as follows: "Takes gr. $\frac{1}{30}$ pilocarpine twice daily, and occasional doses of infusion of digitalis; urine more abundant, its specific gravity 1014, slightly albuminous and containing very few granular casts and blood corpuscles; has lost its smoky appearance; reaction alkaline; perspiration slight; patient convalescent."

Other similar cases might be related, sufficient, I think, to show that pilocarpine given as in above cases in moderate doses, and with sufficient interval, does not produce any deleterious effect and is a very useful remedy in scarlatinous nephritis. It is a remedy, however, that should be given cautiously, at sufficiently long intervals, and discontinued when the urgent symptoms are relieved. I have seen, in one instance, fatal bronchorrhœa rapidly develop in a child that had taken this remedy in too large or too frequent doses.

In the treatment of scarlatinous nephritis, laxatives or purgatives of a hydragogue nature are required, especially when dropsy occurs or symptoms indicating uræmic poisoning are present. Their action is more certain than that of most diaphoretics and diuretics, and their employment is imperatively required in severe or dangerous cases, in which it is necessary to remove as soon as possible the serum or urea, which endangers life.

Young children, or those with delicate stomachs, and those much enfeebled by the primary disease, may take magnesia, either the citrate or the calcined. A good cathartic for ordinary cases is a mixture of jalap and potassium bitartrate, the pulvis jalapæ compositus, consisting of one part of jalap and two of cream of tartar. Ten grains of the mixture may be given to a child of five years, and repeated in two to four hours, according to circumstances. Its effect is increased by dissolving a teaspoonful of potassium bitartrate in a gobletful or half a gobletful of water, and allowing the patient to drink from it. The following cathartic also acts promptly and beneficially in the treatment of scarlatinal nephritis:

℞. Ol. cinnamonii, gtt. viij ;
Magnes. sulphat., ℥i ;
Potass. bitartrat., ℥ii.
M.

Dose.—One teaspoonful repeated from two to four hours, until catharsis occurs.

After the use of laxative agents, the kidneys, being less congested on account of the diversion that has occurred, often begin to excrete urine more freely. But if the patient be anæmic or enfeebled, and the symptoms due to the nephritis be not urgent, it is frequently better to avoid active catharsis, which reduces the strength more or less, and employ remedies of a sustaining character, as in the following case attended by myself :

A boy, pallid and scrofulous, began to have anasarca after scarlet fever, chiefly in the scrotum, with a moderate degree of ascites. The urine, which was passed in nearly the normal quantity, contained albumen, but not in large amount. The patient gradually and fully recovered with no treatment except the use of iron internally and the application of the oil-silk jacket over the kidneys and abdomen to produce diaphoresis. Such a patient, treated by the powerful eliminatives which we employ for the more urgent and robust cases, would probably have been injured rather than benefited. No treatment can, therefore, be recommended for scarlatinal nephritis which will be strictly applicable to all cases. Variations are demanded according to the state of the patient and the form and gravity of the disease.

Diuretics which do not stimulate the kidneys are proper at an early as well as late period of the renal malady. Digitalis has been largely employed in scarlatinal nephritis. I do not hesitate to prescribe it from the first day in ordinary cases.

One teaspoonful of the infusion of digitalis in combination with the acetate of potassium, may be given every third hour to a child of five years. The following formula is for one of this age in good general condition :

℞. Potassii acetatis, ℥ss ;
Infus. digitalis, f. ℥vj.
M.

Dose : one teaspoonful in water.

Local treatment should be prescribed, since it furnishes important aid in subduing the nephritis. Thomas, Romberg, and others, have recommended the application of leeches over the kidneys. Thomas says: "In many cases the abstraction of blood causes immediate and permanent relief; the fever and the pain in the region of the kidneys cease, the secretion of urine becomes augmented, the albuminuria lessens from day to day, and the moderate degree of dropsy that has been developed, disappears." Abstration of blood by leeches or otherwise, I have never employed, and it is likely, in my opinion, to do harm rather than good, unless, perchance, it may be of service in exceptional instances in robust children, who have not been perceptibly reduced by the primary disease. In the majority of cases, instead of depletion, a poultice slightly irritating between two pieces of muslin should, I think, be constantly worn over the kidneys as long as the albuminuria or nephritic symptoms are pronounced. A poultice of equal parts of pulverized flaxseed and ginger, or of one part of pulverized mustard and sixteen of flaxseed, mixed with sufficient water to moisten thoroughly the cloth which retains it, is useful for this purpose. Older children not likely to be frightened by the process, may be treated by the daily application of dry cups over the kidneys, while the body is warmly covered. In subacute cases, not attended by any alarming symptoms, sufficient redness may be produced over the kidneys by one of the mildly irritating plasters constantly worn, which the shops contain.

CHRONIC NEPHRITIS IN CHILDREN AND YOUNG ADULTS.

BY HENRY JACKSON, M.D.,

Physician to Out-Patients of Boston City Hospital.

I DESIRE to call the attention of the Society to the subject of chronic Bright's disease, chronic nephritis, as it occurs in children and young adults. It has been my fortune to observe several such cases, and I feel that the disease may be of more common occurrence than reference to the principal text-book on diseases of children would lead one to suppose. In many of the text-books no mention is made of chronic nephritis. Thomas in Gerhardt's Handbuch speaks of chronic parenchymatous nephritis as occurring occasionally, of interstitial nephritis as very rare; he ends his article on chronic nephritis with saying: "With these few words I leave a subject which hardly belongs in a lecture on the diseases of children."

Biedert in his edition of Vogel's book on diseases of children says, that Vogel never saw but one case of chronic nephritis in a child, though he added in a note of his own insertion, that the disease may occur in children. Baginsky remarks that very little has been written on chronic nephritis in children, and thinks this disease is far more common than is usually considered; he mentions the occasional occurrence of intestinal nephritis. With the exception of Goodhart's most exhaustive article on chronic nephritis in the recent Cyclopædia of the Diseases of Children, Baginsky's article is the only one I have found which would lead the practitioner to consider chronic Bright's disease in children otherwise than as a rarity which might occasionally occur. In fact, in most text-books we find under the heading "Kidney" only acute nephritis as found in scarlet fever, diphtheria and other acute infectious diseases. Dr. L. Emmett Holt, has added an

important chapter to our knowledge of the disease of the kidney in children, by drawing attention to the not infrequent occurrence of primary renal disease in infants, where no historical evidence is forthcoming to suggest the existence of any preceding infection of scarlet fever, or other acute infectious disease. [ARCHIVES OF PEDIATRICS, v. 4, p. 1.]

Henoch says that when chronic nephritis occurs in children the symptoms vary in nowise from those of a similar disease in adults. When we consider that Bright's disease is often one of the most insidious of the diseases of adult life, it seems desirable that more attention should be given to the possibility of the disease occurring in children. Furthermore a careful study of a considerable number of cases in children, may lead to some points of value as to the history or diagnosis of chronic Bright's disease as it appears in children.

Goodhart in his article on Bright's disease in the Cyclopædia of the Disease of Children, divides the disease into two groups: Acute, in which he also places subacute form, and the variety usually described clinically as chronic parenchymatous nephritis. Chronic, applied only to chronic interstitial nephritis. I intend to describe under the title chronic nephritis the forms usually clinically described as chronic parenchymatous and chronic interstitial nephritis. Pathologically these forms are nearly allied, and run one into the other by imperceptible gradation. Throwing out of consideration the question whether the granular kidney may at times be but a later stage of the large white kidney, we certainly have well-marked clinical phenomena,especially prominent as the one or the other of the essential pathological factors predominate. Pathologically a simple parenchymatous disease is found only in connection with systemic poisoning, as by arsenic or some other acute infectious disease. The so-called large white kidney of chronic parenchymatous nephritis always presents more or less evidence of interstitial inflammatory growth. Furthermore, it is very difficult to make an exact diagnosis as to the pathological condition of

the kidneys by a study of the clinical history and examination of the renal epithelium as found in the sediment of the urine. Two cases occurred in private practice; in fact I have for several years made many very careful examinations of the same, both as to the amount of urea and urine passed, and as to the microscopic character of the sediment. (I must apologize for introducing these cases here as I have published notes on both cases in another connection.—*Boston Medical and Surgical Journal*, v. cxxiv. p. 477.)

CASE I.—Boy, fourteen years of age. As a child he had pertussis, variola, measles, and at three years of age a severe attack of tonsillitis. When he was twelve years of age he had in October, 1888, an acute feverish attack, ushered in by chill; for several days he had high fever, and subsequently moderate fever for four weeks; at this time the chief symptoms were diarrhœa and weakness; he had no vomiting and no œdema. He never had scarlet fever and there was no reason to suppose that the acute attack was subsequent to a mild and unobserved scarlet fever. At this time the urine was scanty, contained a large amount of blood, and the sediment was characteristic of acute nephritis. No exposure to wet or cold was noted. Apparently his disease was an acute idiopathic nephritis. In November 1888, examination of the urine gave the following result: Color, yellow, specific gravity, 10.22; a large trace of albumen; urea increased in amount; amount of urine about normal. The sediment contained numerous hyaline and finely granular casts; a good many blood casts; a little free blood. At this time he was rather pale, did not feel strong, but had a good appetite; otherwise no symptoms noted. A month later the blood casts had disappeared from the urine, though the sediment still contained a good many free red blood globules; there were hyaline and granular casts in abundance. There had been a marked improvement in his general health.

In January, 1889, many of the casts contained a few fat drops; the amount of albumen was small. In May, 1889, his mother said he seemed well in all respects; there was no œdema. Examination of the urine gave the following result: Color pale; specific gravity 1015; albumen, a large trace. Sediment contained numerous red blood globules, hyaline and finely granular casts in abundance; on many of the casts a few fat drops. The amount of the urine was

increased, and it contained 20 grammes of urea to the liter. During the last year the amount of albumen has increased to about 1-4 per cent.; the urine has never been less than twenty grammes to the liter; the sediment has shown constantly numerous hyaline and finely granular casts, with fat drops in many of the casts; usually a few and occasionally many free red globules have been found in the urine.

The boy's general health has been perfectly good. No œdema, no symptoms of disturbed nutrition or digestion. No symptoms, except the physical examination of the urine have pointed to the existence of any renal disease. He is small in stature, but for a boy of his age is a good athlete; there is no enlargement of the heart. The fat casts point to chronic degeneration of the parenchyma of the kidney, but the small amount of albumen, the rather large amount of water, and the entire absence of œdema suggest that there is more disease of the interstitial tissue than of the parenchyma of the kidney. There is a constant recurrence of the presence of red blood globules in the sediment, and occasionally a few blood casts appear. On July 31, 1891, nearly three years since the primary attack, I made the following entries of the condition of the urine: Pale color; specific gravity 1076; albumen, one-fourth per cent. In the sediment, very numerous hyaline and finely granular casts ; several fatty casts ; a few blood casts ; a few fatty degenerated renal epithelial cells; an occasional red blood globule. Urea 25 grammes to the liter. Amount of urine increased.

The long continuance of fat casts and fatty degenerated epithelum demonstrate the existence of a chronic parenchymatous nephritis; the absence of œdema, the small amount of the albumen and the rather large amount of urine passed, suggest that the chief lesion exists as a chronic interstitial nephritis.

CASE II.—A young man of nineteen years. He always considered himself well until the spring of 1889 when he was seventeen years of age. At that time, and during the summer he found that he was easily tired, did not feel able to do his usual work, and in general, was "run down." An examination of the urine in September, 1889, showed one-fourth per cent. of albumen; the amount was large, specific gravity 1016, and the sediment contained a good many hyaline and granular casts with a few red blood globules. He had no headache, no vomiting, no œdema; his color was good. No enlargement or other change was found in the condition of the heart.

In November 1889 the examination of the urine was practically the same, except that some of the casts contained fat and a few fatty degenerated epithelial cells were found.

During January and February, 1890, he was in the South at Aiken, and a specimen of urine passed on February 11, showed a small trace of albumen; no casts were found in the urine. On his return in the spring of 1890 the urine again contained albumen and a good many casts. In July, 1890, arsenic was found in his urine, and examination of the wall paper of his bedroom showed that it contained a good deal of arsenic.

During the last year his urine has been examined many times; it has always shown the presence of a small amount of albumen, and usually a few hyaline and granular casts. The last examination in June, 1891, gave a very small trace of albumen; no casts. As his general condition has improved in the last year, and the examination of the urine suggests a gradual improvement in the condition of the kidneys it is, I think, fair to presume that the albumen and casts may have been due to a fatty degeneration of the parenchyma, dependent upon chronic arsenic poisoning. Etiologically the case is of importance, as it would suggest the possibility of arsenic as the cause of some cases of chronic nephritis. I have introduced the case as the first symptoms appeared, when the lad was seventeen years of age, and the examination of the urine pointed to a condition of the kidney which had then already existed for some months, if not years.

CASE III.—A boy, seventeen years of age, entered the Boston City Hospital, May 27, 1884. The previous health had been good; no history of scarlet fever or diphtheria; no marked exposure to cold. Nine days before entrance to the hospital he had pain in the stomach with some vomiting; slight headache and "feverishness"; these symptoms were soon followed by general swelling. On entrance he had considerable œdema of the legs and scrotum; slight puffiness of the face; some cough and dyspnœa on exertion.

The urine was pale, acid, of specific gravity of 1020; one-fourth per cent. of albumen. In the sediment, numerous coarse and fine granular casts were found, also free fat and a few fatty casts.

During a stay of two months in the hospital, his general condition improved markedly, and the œdema almost disappeared. There was no marked change in the character

of the urine. Here again, the examination of the urine and the subsequent course of the case indicate that the disturbance on account of which he entered the hospital, was but an acute intercurrent exacerbation in a chronic nephritis.

CASE IV.—A boy sixteen years, of age, a plumber, entered the Boston City Hospital, April 2, 1885. He was well until he was fourteen years of age. During the two years previous to entering the hospital, he had numerous attacks of severe and persistent vomiting, accompanied by frontal and occipital headache; he has had two convulsions; no swelling noticed.

On his entrance to the hospital he had obstinate and frequent vomiting, severe headache; no œdema. The area of cardiac dulness was not increased; a short soft murmur was heard just beneath the nipple.

An examination of the urine showed it to be pale in color, large trace of albumen, and in the sediment an occasional hyaline and fine granular cast was found; the specific gravity was 1011

A month later he had less vomiting, was able to sit up; the urine showed the same characteristics; the amount of urea passed was eleven grammes to the liter. Ophthalmoscopic examination showed a slight haziness of the optic discs.

He left the hospital in May and was able to work for a few weeks, but reentered the hospital on the 27th of June. At that time he had again much vomiting and frontal headache; on the following day two uræmic convulsions.

In July slight œdema of the legs developed; an examination of the heart showed that the area of cardiac dulness was increased.

Throughout the summer he had excessive vomiting, with severe headache; constant examination of the urine showed that it was of low specific gravity, contained a small amount of albumen, and in the sediment there were a few hyaline and granular casts. On September 10, he had a convulsion and died suddenly.

The autopsy showed interstitial nephritis, chronic pachymeningitis, hypertrophy of the left ventricle of the heart, œdema of the lungs, and chronic adhesive pleurisy.

In this case we have a typical history of chronic interstitial nephritis, ending fatally in a lad of sixteen years of age, which had existed certainly for two years, and probably for many more years, as for two years he had symptoms of uræmic poisoning.

CASE V.—A girl of thirteen years entered the Boston City Hospital, June 18, 1887. When an infant she had diphtheria; otherwise no illness. Three weeks before entrance she had nausea, no headache or vomiting; there was some œdema of the legs. On entrance she felt well, her only complaint being œdema of face, abdomen and legs. The urine was pale, of specific gravity of 1020; one-fourth per cent. of albumen; in the sediment numerous hyaline and fine granular casts, some of the casts containing fat. After remaining in the hospital for a month, she left against advice without showing any improvement.

CASE VI.—A boy, seven years of age, entered the Boston City Hospital, September 1, 1887. He never had scarlet fever or diphtheria. On entrance there was general œdema; pulmonic second sound was accentuated. The urine was yellow, of specific gravity of 1020; it contained about one-half per cent. of albumen; in the sediment were numerous hyaline and granular casts, some blood, and a few fatty casts. During a stay of four months in the hospital, there was a marked diminution in the œdema, and improvement in his general health. There was no change in the character of the urine.

CASE VII.—A boy, eight years of age, entered the Boston City Hospital, June 24, 1891. For five weeks before entrance he had general œdema of face, legs and abdomen; during the whole of the time he was confined to the bed. The urine was brown in color, specific gravity 1030; two to three per cent. of albumen. In the sediment were numerous hyaline and granular casts, ephithelial, blood and fibrinous casts.

Face puffy, general œdema of the extremities and marked ascites. Heart negative. One week later the urine was pale in color, specific gravity 1017; one per cent. of albumen; in the sediment were numerous fatty elements.

During July occasional vomiting; great discomfort from ascites; œdema of the legs and scrotum marked.

On July 6, the urine contained two per cent. of albumen and there was a good deal of blood in the sediment. During July and August there was much oedema of the legs and scrotum, with ascites sufficient in amount to produce a great deal of discomfort from pressure. No vomiting or headache.

On August 20, the ascites was so marked that it was considered advisable to tap the abdomen and a large

amount of fluid was drawn off, about four-fifths of a pailful. This operation was immediately followed by great relief of all the untoward symptoms; the urine increased in amount and the œdema of the extremities rapidly decreased. An examination of the urine August 27th, showed a pale color, specific gravity of 1010; albumen, slight trace; some blood; no casts found. During September his general condition has been good; the amount of albumen in the urine has varied, and the sediment has shown hyaline and granular casts, some fat casts, and at times, blood.

The ascites has not returned; there is slight œdema of the legs, not always present. No enlargement of the heart; no murmur.

CASE VIII.—In August, a boy of fifteen years, presented himself at the out-patient department of the Boston City Hospital. When two years of age he had scarlet fever; his general health was good, but not robust. His mother said that for several months he had been failing in strength; his appetite was fair; occasionally he had slight headache and some vomiting. Physical examination showed a "pigeon-breast" deformity of the sternum; his heart was enlarged and the second pulmonic sound was much increased; apex in line of nipple; no murmur. Examination of the urine: Specific gravity 1018; one-fourth per cent. of albumen, and the sediment contained a few hyaline and fine granular casts with an occasional epithelial cast and a few red blood globules. He was somewhat anæmic. A month later, after passing four weeks at the seashore, and taking iron he felt stronger. An examination of the urine was practically the same, except that the albumen was less in amount.

The enlargement of the heart and the presence of casts in the sediment in this case, are sufficient to remove the case from the category of cases classified as the "albuminuria of adolescence." The fundum of the eye was normal. No œdema of extremities. Some of these cases may entirely recover from the lesion of the kidney as demonstrated at the time of the examination of the urine, but I consider them worthy of mention as calling attention to the fact that a chronic disease of the kidneys may be present in children as in adults, and yet not always give rise to symptoms suggestive of renal disease unless

the urine is examined. In the last case mentioned the hypertrophy of the heart alone suggested the possibility of renal disease.

I am indebted to the staff physicians of the Boston City Hospital for permission to report the cases which entered that hospital.

TWO CASES OF ACUTE PRIMARY NEPHRITIS IN INFANCY.

BY L. EMMETT HOLT, M.D.,

New York.

ACUTE primary nephritis in infancy is certainly not a common disease. The two cases here reported are submitted to this Society with the hope that they may shed some light upon this obscure subject, which has as yet received so little attention. Following that strange law of coincidence so frequently seen in medicine, both cases came under observation within a few months of each other.

The diagnosis in the first case was overlooked during life; while in the second case it was readily made from its resemblance to the first.

CASE I.—A female child, six months old, was a patient in the Babies' Hospital. On admission it was stated that the child had been well until about six days before. It had been nursed by the mother up to that time, when it was taken with what was apparently an ordinary attack of indigestion, frequent vomiting, and fever. The symptoms continuing for three days, the infant was weaned by the advice of a physician, and a diet of condensed milk substituted. No improvement followed this change, and as the symptoms gradually grew worse the child was brought to the hospital,

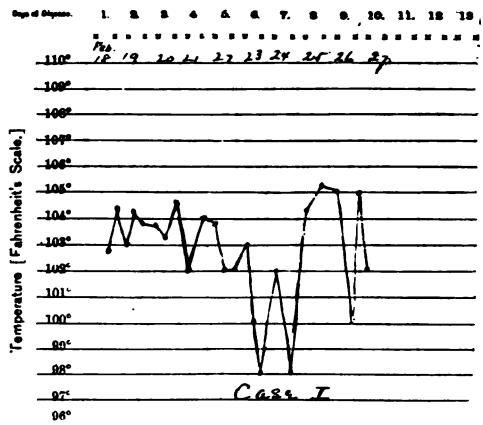
She had never to the mother's knowledge had any of the infectious diseases.

On admission, February 18th, the general nutrition was good, the child being plump, well nourished, weight 13½

pounds. The fontanel was widely open and there were several small spots of cranio-tabes to be felt over the skull.

The tongue was dry, but mucous membranes of mouth, nose, and pharynx were normal ; skin likewise normal ; no enlargement of external lymphatic glands ; spleen and liver normal in size ; heart normal ; a few coarse râles over both lungs behind. There was a good deal of prostration, the infant appearing to be seriously ill ; temperature, 102.8° F., pulse and respiration both rapid but regular.

During the first three days in the hospital, February 18th, 19th and 20th, there was increasing prostration with a dull apathetic mental condition. The infant coughed occasionally, but daily examination of the lungs revealed



only a few coarse moist râles. There was some dyspnœa, the alæ nasi were active and the respiration at times rose to 76 per minute. The temperature did not fall below 103° and ranged between this point and 104.5°. The vomiting soon ceased and the child took twenty ounces daily of a dilute milk and cream mixture.

The case was regarded asone of probable pneumonia.

During the next three days, February 21st, 22d and 23d, the signs in the chest were rather less marked, the respiration ranged from 40 to 50 per minute, and the pulse about 150. The temperature still remained elevated, but fluctuated irregularly, usually between 102° and 104°, once dropping to normal. No change in the general symptoms, except that the child was weaker and extremely

pale ; still dull and apathetic with no other pronounced cerebral symptoms.

A distinct remission of the temperature upon February 23d and February 24th led to the suspicion of malaria, and the spleen now appeared to be slightly enlarged, as it was distinctly felt below the ribs.

Quinine was accordingly tried in full doses, but without any effect. The pulmonary signs were still very few and inconstant, the respirations ranged from 38 to 40 per minute, and the pulse about 150.

On February 25th and February 26th the strength failed steadily and the anæmia increased. The face had now assumed the waxy look so resembling that of an old case of nephritis, and attempts were made to collect urine for examination, but these were unsuccessful as the bowels moved almost every time water was passed. There was no œdema, and the urine had not been noticeably scanty during the illness.

The temperature during the last three days fluctuated irregularly, nearly reaching 105° every day. The child's pupils were small but usually responded to light ; once nystagmus was noticed.

She sank gradually and died of exhaustion on the morning of February 27th in the eleventh day of her stay in the hospital and upon the seventeenth day of her illness.

Let us briefly review the symptoms ; they were :

(1) Continuous fever of an irregular type, but generally high.

(2) Dulness and apathy with no focal symptoms of brain disease.

(3) Marked anæmia, without emaciation.

(4) Moderate gastro-intestinal disturbance, marked in the early part by vomiting, and late by loose evacuations from the bowels.

(5) Evidences in the lungs of a moderate amount of bronchitis of the larger tubes.

The obscurity of these symptoms is so evident that I will not dwell upon them, but give the results of the autopsy, without which we must have been left entirely in the dark regarding their explanation.

The *autopsy* was held fourteen hours after death. Body well nourished.

Head.—Meninges œdematous. Brain sinuses empty. Brain substance normal.

Chest.—A few small nodules of broncho-pneumonia in both lower lobes behind and superficially situated, the

largest being less than one-half inch in diameter. Six or eight hæmorrhagic infarctions in the lower lobes, the largest one being three-quarters of an inch in diameter. The lungs, posteriorly, showed moderate congestion and were everywhere œdematous. There was a slight amount of general bronchitis.

The heart was normal.

Abdomen: Spleen normal. Liver pale and slightly fatty; size normal. Stomach normal and empty. Small intestine contained healthy fæcal matter, and its mucous membrane was normal, as were Peyer's patches.

The colon showed no recent changes, but there was quite an unusual amount of swelling in the solitary lymph nodules.

Mesenteric glands slightly enlarged.

The kidneys were decidedly enlarged, fully one-third larger than normal. They were unusually soft. The surface of a grayish-yellow color, mottled with red. Capsules not adherent. On section, the cortex was slightly thickened, of a grayish-yellow color, mottled in places with red, and here and there were wedge-shaped areas of a uniformly yellow color; pyramids very yellow in color and markings indistinct.

Microscopical examination of the kidneys by Dr. Martha Wollstein, Assistant at the Laboratory of the Woman's Medical College: "The cells lining the capsules of the glomeruli are proliferated, and those covering the tufts are so to a very marked degree. The epithelium lining the tubules is everywhere swollen and granular, and in some places peeled off. The lumen of the tubules is filled with exudative products, granular casts. The stroma is markedly infiltrated with small, round cells; in the neighborhood of the small blood vessels these are especially crowded. The blood vessels are congested. The wedge-shaped areas seen by naked eye are made up of infiltrations of small round cells. The anatomical diagnosis is *acute diffuse nephritis*."

CASE II.—Female child, thirteen months old, an inmate of New York Infant Asylum for eight months. Nursed by mother and well nourished, weighing 18½ pounds at beginning of attack. Never had measles, scarlet fever or diphtheria; taken suddenly ill, without any assignable cause, on June 12th, with temperature 103.8°.

On examination, throat and chest negative, spleen not felt; child rather dull, several molars nearly through; bowels not moved during the day.

During next three days, June 13th, 14th and 15th, the bowels moved from two to three times daily; the temperature continued high, from 101° to 105°. A few coarse râles were found in lungs, physical examination otherwise negative; rather restless; nursing well.

On June 16th, 17th and 18th, there was occasional cough, increasing prostration, and pallor noted; no further signs in chest; slight diarrhœa; no vomiting; pulse 120 to 140; respiration, 40 to 60. Temperature still high, from 102° to 105°, partly controlled by baths. Bowels continued rather loose, from two to four movements a day. Two molars came through on the 17th.

On June 19th, 20th and 21st, there were from one to two passages daily from the bowels, yellow and thin in character, still no vomiting; child growing steadily weaker, and pallor more striking. She now became more irritable and restless; occasionally there was twitching of the facial muscles; these symptoms alternated with drowsiness. Pupils normal; no rigidity of neck. Pulse and respiration regular, frequency about as before. Temperature up to 105° every day, and once 106°. Chest still continues negative; has cut two more teeth.

June 22d, 23d and 24th.—Tongue dry; condition more apathetic; quiet most of the time; irritable and cries if disturbed; pupils at times irregular, and respond feebly; pallor extreme; respiration 40 to 50; pulse 140 to 150, and weaker.

June 25th, 26th and 27th.—General condition practically unchanged; at times quite bright, but greater part of the time dull. Pulse 150 to 180, and weaker; respiration still regular. Temperature a little lower, 101° to 104°. One to four thin movements daily.

June 28th and 29th.—Passages rather more frequent, four to five daily. Slight cough; lungs still negative on physical examination; increasing prostration; nourishment taken poorly.

On June 30th I saw case for first time. I found a plump well-nourished infant, with no signs of emaciation, but with extreme pallor; the skin had a waxy look; lips almost bloodless; fontanel normal; no rigidity of neck; a moderate amount of thrush in mouth; throat negative, and also the lungs, with the exception of a very few coarse râles behind; heart normal; spleen felt just at edge of ribs; liver one inch below ribs; abdomen natural: no retraction, no tympanites, no eruption; a moderate amount of dyspnœa, and at times a slight amount of cyanosis.

There was a very slight amount of swelling of the feet and ankles, but no puffiness under the eyes. Slight muscular twitchings of left leg.

The infant had now been ill nineteen days, during whole of which time the temperature had been steadily high, touching 105° almost every day, and usually fluctuating between 101° and 105°. Pulse gradually becoming more rapid and weaker; respiration accelerated but not in proportion to the rise of temperature. In addition, slight diarrhœa most of the time, averaging from three to four movements a day. At no time any vomiting; the nervous symptoms slightly more marked during last four or five days, but not at all characteristic; added to this only a marked amount of anæmia, without emaciation. The family history was negative, and the examination justified us in excluding meningitis, malaria, and typhoid fever. It seemed impossible that the intestinal disturbance could be the cause of the fever and the anæmia, because of the absence of emaciation. The urine had been passed freely, it was stated, during the whole attack, and an examination made early in the illness showed nothing abnormal.

The resemblance of the case to the one first reported, particularly the fever and the anæmia without evident explanation, led me to suspect acute nephritis in this one. An examination of urine showed about three per cent. albumen by bulk; under the microscope, pus cells in quite large numbers, epithelial and granular casts, and renal epithelium, all being very abundant; a few red blood globules. An examination of the blood showed the changes of ordinary anæmia only.

During two following days there was no essential change in symptoms except gradually increasing prostration and drowsiness; some difficulty apparently in micturition, but the water seemed to be passed freely; it was impossible to collect enough to form any idea of the daily quantity.

A second examination showed essentially the same urinary conditions. Death occurred from exhaustion, July 2d, on the twenty-first day of illness.

Autopsy.—Twenty hours after death. Body well nourished. Cerebro-spinal fluid slightly in excess of the normal. A non-adherent, partly decolorized thrombus in superior longitudinal sinus. Brain and meninges in other respects normal.

Lungs anteriorly pale and anæmic, posteriorly a few small spots of broncho-pneumonia. Pleura normal.

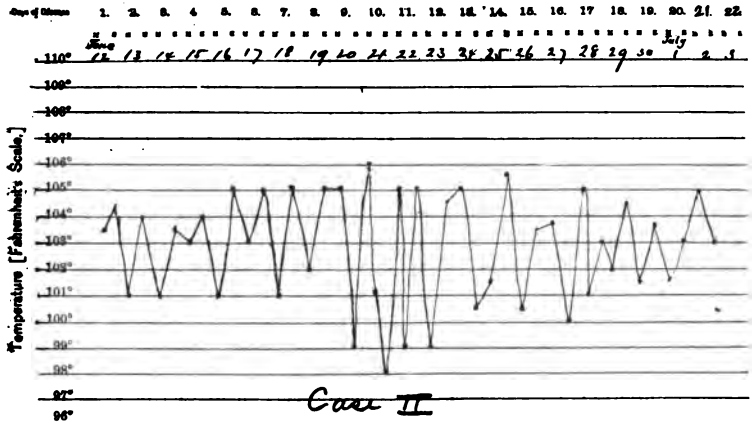
Heart very pale and walls slightly fatty, otherwise normal.

Stomach pale, but in other respects healthy. The small intestine contained thin fæcal material. The mucous membrane of the ileum was pale, that of the jejunum bile-stained ; no evidences of enteritis.

The colon contained thin greenish matter but the mucous membrane was pale.

Liver normal in size, but very pale. Spleen slightly enlarged, also paler than normal. Pancreas normal.

Kidneys much enlarged; weight, 3¾ ounces. They were very soft, capsules non-adherent, surface of a yellowish-gray color, with a fine red mottling. There were many small circumscribed areas which were very yellow,



these varying in size from a pin's head to a fourth of an inch in diameter. On section the cortex was thickened, normal markings almost obliterated ; color like that of the surface. Pyramids of a yellow color streaked with red. The yellow areas of the surface extend into the organ as small wedge-shaped masses.

Microscopical examination of this case was made by Dr. Eugene Hodenpyl, Assistant at the Laboratory of the College of Physicians and Surgeons. The lesions were essentially the same as those described in Case I., with the exception that the changes in the glomeruli were not so well marked, while the areas of round-cell infiltration were larger and more numerous. The anatomical diagnosis was the same, *acute diffuse nephritis.*

In 1887 I published (ARCHIVES OF PEDIATRICS, Vol. iv., pp. 1 and 103) a series of six cases of acute nephritis, and collected from medical literature enough to make twenty-two cases, all that I could find recorded at that time. Of these six cases, three, Cases I., II. and IV. were in all respects similar to the cases reported in this paper, and the diagnosis was confirmed by autopsy and microscopical examination of the kidneys in two of the cases. Since the publication of that paper I have not met with a case until those whose histories have been here detailed. A closer examination of the records of these five cases reveals a very striking resemblance between them. They were all between the ages of six and thirteen months. There was in all a prolonged febrile attack; this lasted in the shortest case seventeen days, in the longest one, twenty-six days. In no case was there marked dropsy; in only one case was the amount of urine noticeably diminished. Nervous symptoms were present in all. The lesions, both gross and microscopical, in the four cases in which autopsies were obtained were strikingly similar, differing only in degree. The records of this group of cases make up a chapter in the literature of diseases of the kidneys which I think has not before been written. They show the great necessity of examinations of the urine in all obscure febrile attacks in children.

DISCUSSION.

Dr. SEIBERT:—In the beautiful case related by Dr. Holt, the question is, where did the infection come from? The frequent occurrence of mild attacks of tonsillitis in infants that are so often overlooked and are described as one-day fevers, if we examine carefully, we find acute tonsillitis. Now there is an entrance, an opportunity for infection. Why could not infection come from this source? I want to call attention to a fact in one of the cases described by Dr. Putnam. The child did not have scarlet fever nor diphtheria, but did have varicella. I had two cases following varicella and we will find this result more frequently than we think. In regard to the chronic cases I think the same is true. We have acute attacks, following some little affection that we have made nothing of and then later in the disease kidney trouble is found. Therapeutics were not discussed, but I would like to mention a new combination. It is called diuretine, a combination of theobromine and salicylic acid. Since the first of January I have made use of this in heart and kidney disorders

and it is the quickest diuretic I have seen. I use it in nephritis following scarlet fever.

Dr. FORCHHEIMER :—It is years ago that I had my attention called to this connection by Prof. Hebra, who may have had a hobby, but he claimed that catching cold did not exist, basing his views on a great number of cases. This catching cold question has done a great deal of harm to us as physicians in connection with scarlatina. The predisposing cause seems to be in the nature of the cause. I have had an epidemic of scarlatina in Cincinnati, lasting eighteen months without a case of nephritis. I have seen epidemics with thirty per cent. of cases of nephritis. I have tested this question of catching cold. Avoid exposure as you will certain ones will and certain ones will not take nephritis. My method has been to take a certain number of cases, treat them by absolute rest, closing the windows, putting up screens, and avoiding exposure to draughts, then taking an equal number to expose them as much as I could, the result was the same in both instances as far as nephritis was concerned. Of course cases in the same experience only can be utilized. I think this is a very fruitful subject for experiment if we go about it in an impartial manner.

Dr. KOPLIK :—I have been working the past year in this line and am interested in the pathology of these cases—the changes which take place in scarlet fever and their exact causes. According to the most advanced researches the cause is microbic. If the microbes which have been isolated in these cases are injected into a small animal you find processes in the kidneys almost the exact counterpart of those which have been described in diphtheria and scarlet fever. Nephritis has been thus produced artificially. There is the necrobiosis and swelling of epithelial cells almost the exact counterpart of the disease in the human subject.

Dr. SMITH :—Will you please tell us what the microbe is?

Dr. KOPLIK :—A streptococcus identical with the streptococcus of Babes. Dr. Prudden has also described these streptococci in the lungs of diphtheritic cases in complicated scarlet fever.

Dr. PUTNAM :—The question is, whether the usual opinion in regard to the cause of nephritis after scarlet fever is exposure or not. The question is whether plenty of fresh air is better than a diminished amount, and we ought not to consider it as anything more than this:

Whether children are to be kept in bed for four weeks after exposure to scarlet fever, or whether they shall be allowed to be up and around. I believe in bathing freely, keeping the air of the room cool and fresh, and not keeping the child in bed all the time, for this is very weakening. I do not want to say that the kidneys cannot be congested by the child's being dipped in cold water. I have never been able to see any evidence that exposure had caused scarlatinal nephritis. It occurs often in the cases best protected. It has been my practice to have children with scarlatina bathed all over once or twice a day.

Dr. KOPLIK:—I am pleased to see that the President has made the point he has. I should be ashamed to have the question left so one-sided; though we think the nephritis is mycotic, we must recognize other predisposing causes also. We must guard our patients against all needless exposure.

Dr. CARR:—The cases we see in our patient departments are those that are called scarlatina, or a mild form of scarlet fever. From the nature of things we see more of these than of the severer types, and the causes of nephritis in these cases are probably exposures and over-exertion rather than simple fresh air. Hospital statistics have shown the lowest percentage of nephritis to be in the patients kept quiet in bed.

Dr. FRUITNIGHT:—In many of the mild cases which are allowed to go about, it is not the cold itself, but the exposure that acts as the exciting cause. Just what the essential systemic cause is, we cannot tell.

Dr. HOLT:—Have any of the gentlemen here met with the purulent form of nephritis?

Dr. NORTHRUP:—I have met with one case. An infant suffered two months and died. No diagnosis was made and at the *post-mortem* its kidney was found full of pus.

Dr. SMITH:—I am somewhat skeptical as to certain cases of nephritis in children reported as primary. On careful examination of cases which seemed to be primary at first, I have now and then found, as Dr. Seibert says, that there has been probably a trivial antecedent diphtheria or scarlatina not detected. A child has been slightly sick with a little redness of the throat, but it was so trivial that the doctor who saw the case for me did not call again. A week later the child had what proved to be a fatal nephritis, which, I have no doubt, was due to a slight diphtheria. In one case of severe nephritis, I was told that the child had been previously well and daily out of

doors until the kidney disease began, and that there was no diphtheria or scarlatina in the vicinity to which it could have been exposed. But I found subsequently on inquiry, that this child and its sister had a rash a few days previously, which was thought to be due to the summer heat. Nevertheless, that there is a primary nephritis in children as in adults cannot be doubted, due probably to taking cold, and cases of primary nephritis reported by so thorough an investigator as Dr. Holt, I am prepared to accept as such.

Dr. JACOBI:—It was a surprise to me to hear that the text books contain nothing about acute nephritis in the very young. No doubt Dr. Holt has looked over our own and the European authorities and knows what they say, I shall look them over myself, for my own satisfaction. A text book cannot be expected to contain a full account of all diseases, for each alone would form a large monograph. Such omission simply proves that this writer was not in the way of seeing such cases. It is a surprise, however, that it is not mentioned, for I have seen twenty or more cases. Wherever we find cases of nervous disturbance, the urine is examined. I have made the diagnosis of primary nephritis in many cases. In a baby of five weeks, in the practice of a well-informed practitioner, a member of this society, who had overlooked this cause of the dangerous symptoms. I am positive that I have seen a few cases of the primary form every year. I never leave my house without a small and medium size catheter, and often have occasion to use them. I have seen a number of these cases in which the diagnosis made during life was confirmed at the autopsy. There are plenty of opportunities for children to have nephritis. Even in the first week of their life they are so threatened by lesions produced by uric acid infarcts and stone. Of course, the causes which affect the infant are not so numerous as in the adult, except the eruptive diseases, but I do believe that exposure to cold does produce nephritis in some cases. I have seen children die within three or four days from nephritis, the result of exposure to wet and cold. There are a number of other cases, for instance, those which complicate intestinal diseases. It is true that many cases of vomiting and diarrhœa are merely symptoms of nephritis. A number of cases supposed to be cholera, even the Asiatic, are found to be acute nephritis. On the other hand, where we have to do with an acute or sub-acute intestinal catarrh, a prolonged seizure may give rise to secondary nephritis. I

am positive that it will be found to be much more frequent than it was considered to be.

Dr. JACOBI:—I want to ask whether Dr. Seibert has responded to the question or not.

Dr. SEIBERT:—Do I permit exposure to draught? Yes; all I can get hold of.

Dr. JACOBI:—We all want fresh air, but Dr. Seibert is inconsiderate. If I open the window, I want a screen between the bed and the window. We want air, but not draught.

Dr. ROTCH:—I wish to protest against what has been said in regard to taking cold. Exposing a child with scarlet fever to cold, is wrong, I believe it is criminal. Perhaps it is not scientific to say catching cold, but something comes from the exposure and scarlatinal nephritis, may, I think, be precipitated by lack of care in the nursing, allowing internal congestions to take place from too rapid and unnecessary surface evaporation.

Dr. SEIBERT:—I am sorry I have committed that outrage so frequently. Among my own relatives scarlet fever has carried off a young man, aged twenty-four in thirty-six hours, his sister, a girl of seventeen, was down with scarlatinal diphtheria, purple cheeks, enormous glandular swelling, high fever, and the other symptoms. The father was down with diphtheria, the mother with scarlet fever and diphtheria. I had seven patients there. I took the stove out of the room, though there was snow on the ground, and had the four windows opened, and put ice to the throats of the patients, and they got well. This, to my mind is the most satisfactory treatment. When allowed, I always treat scarlet fever this way. The worse the case, the cooler the room. I would not roll a naked child in the snow, but there is sense in forbidding pure, cold air, in an infectious disease, where the cold air itself is the best stimulant we can get.

ABSCESS OF LUNG.

BY FRANCIS HUBER, M.D.,

New York.

Benny J., four years of age, pale and emaciated; presented the appearance of a child suffering with a long-continued suppurative process at the time he came under my observation in October, 1888. The history in brief was, that he had been sick about five weeks with a pleuro-pneumonia. During his illness, he had lost considerable flesh and became pale and waxy-looking. Fever present throughout the course of the disease; at the outset the temperature was continuous, later on varied considerably —sometimes high, at other times but moderate. Irregular chills had been observed, with profuse sweating now and then. The cough was of a distressing character, not attended with much expectoration. Dyspnœa considerable; diarrhœa at times.

Physical examination revealed flatness on the right side in the infra-clavicular and mammary regions. Axilla and posterior portions normal. Vocal fremitus absent over the area of flatness. Auscultating, find an absence of vesicular breathing and voice during quiet respiration. Deep inspiration or coughing elicited faint and distant bronchial breathing. Posteriorly on the right side a few coarse râles discovered. Left side of chest normal. Heart's action regular, somewhat accelerated; apex beat not displaced. Liver not displaced downwards.

Diagnosis.—Localized empyema of the right side anteriorly, corresponding to the infra-clavicular and mammary regions.

October 31st.—In view of the interesting nature of the case, the child was presented at the Vanderbilt Clinic. Prof. A. Jacobi confirmed the existence of the above-mentioned physical signs and suggested the possibility of the existence of an intra-pulmonary abscess. An exploratory puncture revealed pus.

November 1st.—Assisted by Dr. Charles E. Denhard and Dr. Margolies, the patient was chloroformed and as a preliminary measure an exploring syringe was again introduced and pus readily found. Certain of my diagnosis of localized empyema, I rather injudiciously withdrew the needle and left myself without a guide. An incision about two inches long was made along the upper border of the fourth rib and the structures were divided layer by layer until the pleura was reached which was then incised, but no pus escaped. A long exploring needle was now passed in in different directions a number of times, finally pus was detected. A grooved director was then introduced alongside the needle as a guide, upon which a pair of dressing forceps were inserted and the opening dilated. Considerable blood mixed with pus escaped. A drainage-tube was introduced and the cavity gently irrigated. Dressing of iodoform-gauze and borated cotton applied.

The operation clearly demonstrated to Dr. Denhard and myself the intra-pulmonary character of the abscess, and showed that Prof. Jacobi's suspicions as to the exact nature of the affection were correct. Adhesions had fortunately taken place between the visceral and parental pleural surfaces, and the abscess was therefore reached without involving the general pleural cavity. The hæmorrhage from the lung upon dilating the opening with the dressing forceps was brisk but not excessive. The abscess was probably situated about half an inch beyond the pleural surface. The main cavity of the abscess was evidently, not incised for two days subsequent to the operation, the discharge was very much more profuse, several ounces of pus being discovered upon the dressings. An extensive pneumonia developed in the meanwhile (bronchial breathing being heard in the axilla and posteriorly above) no doubt traumatic in character, due to punctures made in the attempt to locate the pus after the incisions had been made and the pleura opened.

Without any difficulty pus was readily detected by exploratory puncture the preceding day and just prior to the incisions. When the pleura was incised, as the landmarks were obliterated, considerable trouble was experienced, and several punctures were made before the pus was

located. Had the precaution been observed to leave the needle in situ the difficulty would not have been met with, nor would the traumatic pneumonia have been so extensive. In a subsequent case I shall certainly leave the needle in as a guide. The pneumonia resolved in a satisfactory manner in about a fortnight. The discharge from the abscess gradually diminished and the patient began to improve rapidly. In irrigating, it was noticed that when a gentle stream was allowed to flow no cough occurred; if however a little force was employed a severe spasmodic paroxysmal cough followed, to cease only when the fluid which had escaped into the bronchi had been expectorated. A further argument in favor of the intra-pulmonary character of the abscess.

January 17, 1889.—The patient was presented at the Academy of Medicine (Section of Surgery). General condition excellent; a little rude breathing at the upper and posterior portion of the right lung; anteriorly, breathing good. A small sinus is still left with a small drainage-tube in situ, secreting about one drachm in four days when the dressings are changed. A forcible injection of fluid still produces the spasmodic cough. The tube was retained for some time longer, and in May the parents moved to better quarters, the patient being taken out daily and allowed to play and enjoy himself in the park. Under the improved hygienic conditions the discharge diminished until but a few drops were found upon the dressings at the end of several days. The tube was now dispensed with, the sinus gradually contracted and closed completely towards the latter part of August, ten months from the onset of the disease.

Purulent collections, the result of tuberculous or metastatic processes, embolic or pyæmic in their nature, or those induced by traumatic influences, or the impaction of foreign bodies in the bronchi are by no means infrequent in the lungs. Simple non-tubercular and non-metastatic abscess on the contrary are but rarely encountered. According to S. Seabury Jones, pulmonary abscesses may be met with in one of four forms.

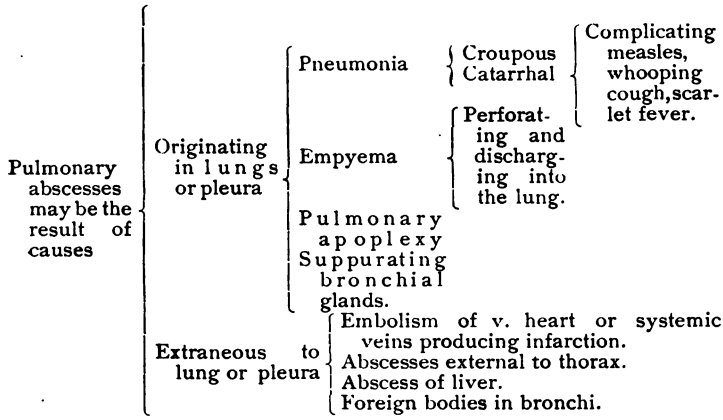
In the first variety the symptoms are very obscure from the beginning and remain so until a discharge of pus takes

place. In the second class the symptoms resemble those of pleurisy with effusion, and in the third those of pulmonary tuberculosis. Finally, the fourth variety is associated with pneumonia, which advances slowly from one lobule to another or is characterized by a tendency to skip from one lung to another.

The affection is occasionally met with in children of weakly constitutions living under poor hygienic and sanitary conditions. The general health in these cases is lowered and debility becomes an important factor in the causation of the disorder. Meigs and Pepper (Diseases of Children, seventh edition, page 162) say "abscesses are not very uncommon in the lobar pneumonias of children. They occur as a result of the third stage of the disease." Other authorities, as Henoch, Gebhardt, Eustace Smith, on the contrary teach that the condition is rather rare. Minot, (Cyclopædia Diseases of Children, vol. ii., page 589) states that suppuration and gangrene are rare in children, only occurring in the severest and usually fatal cases.

Ashby and Wright devote less than twelve lines to the subject. They claim that the abscesses are mostly the result of septic embolism from some distant suppurating centre, as in an otitis or some other bone lesion, and associated with pyæmia. They are usually small and situated on the surface. Small abscesses may be secondary to an empyema, the latter finding its way via a small abscess into the bronchial tube. Minute abscesses are sometimes a sequence of a broncho-pneumonia secondary to scarlet fever, measles or whooping cough, suppuration taking place in the lobules immediately surrounding the terminal bronchioles.

Delafield remarks, "that the inflammatory processes may be so severe in rare cases, and cause such compression of vessels from excessive exudation of fibrin that death of small portions of the lung tissue results. Then necrotic portions become separated from the surrounding lung, which is infiltated with pus, and in this way abscesses are formed."



In children as well as in adults, the process may be met with in the insane. Such a case was under my observation a few years ago. Sammy L., æt. eleven years ; very childish in his way with occasional brief maniacal attacks, had been sick for several weeks with symptoms of an irregular typhoidal character. Hectic fever with irregular chills present. The boy emaciated rapidly, coughed a good deal but did not raise much. Dyspnœa quite marked, reminding one of effusion. Physical signs negative with the exception of dulness of posterior upper portion of right lung with a few râles. At one of my visits he was seized with a severe paroxysm of coughing, during which some pus, quite offensive, was expectorated. As the patient was rather intractable, a very careful physical examination was not possible. He began to improve after this, and was soon able to go about. He then passed from under my observation, being advised to go to a mountainous district. After a few months he returned to the city fully restored to health as far as his pulmonary trouble was concerned, the cough having disappeared, his general physical health about as usual. He was still in good health when heard from a short time ago.

The old writers remark: "When after a pneumonia an abscess forms, there is fever, dry cough, dyspnœa, swelling of the feet." This picture may be true in advanced cases or large abscesses. The recognition of the complication at the onset is obscured or rendered difficult of correct interpretation by the presence of symptoms which preceded the suppuration. When the latter has been

fully established we may have the characteristic evidence of pus formation. In the simplest and most frequent clinical form, that which follows a pneumonia, a history of rigors, sweating and increased vital depression with the temperature variations, should direct our attention to the possible existence of this complication, though even now we may not be able to localize its exact position.

The temperature may be high, or if it falls, rises rapidly and assumes a hectic type. There is usually great weakness, the tongue becomes dry and brown, the complexion dull and earthy-looking with perhaps lividity of the eyelids and lips. Our suspicions may be aroused or verified by a sudden expectoration of pus, more or less copious, followed by the physical signs of a cavity at a circumscribed portion of the lung, previously in an infiltrated condition.

It is probable, though no careful examination of the sputum of children suffering from the disease has been reported, that particles of lung tissue, pus corpuscle, and crystals (fat and hæmatoidin) described by Leyden, as occurring in adults, occur under the same conditions at an earlier age. If the abscess be small and deeply seated, or if multiple and disseminated, the signs may be obscure or wanting. If in close proximity, the smaller abscesses may coalesce to form a larger one. Dr. L. E. Holt (*Medical Record*, April 16, 1887) reports an extremely interesting case in a little girl but three months old. The child lived twenty-four days after the onset of the trouble, and at the autopsy the right lower lobe was found to have been converted into an abscess sac with thin walls, the cavity holding about four ounces. In the *Medical Bulletin*, Philadelphia, September 13, 1890, D. Maclean presents a case of suppuration, with destruction of the entire left lung, treated by resection of ribs and drainage of cavity, followed by recovery.

In the fatal cases of broncho-pneumonia following whooping cough, measles, and scarlet fever, numerous yellow spots, the size of a millet seed or larger are scattered throughout the lungs, "graines jaunes," or abscès

peribronchite of French authors. They are minute abscesses surrounding the terminal bronchioles formed by the softening of the pneumonic lobules.

The suppurative process may run an acute course or extend over a period of months. It may occur coincidently with the original disease, or may not manifest itself until a later period, the lung tissue having remained partially consolidated. In some instances the walls are sharply defined, in others there seems to be no limiting zone, the suppurating process apparently being gradually lost, or rather becoming blended with the normal pulmonary structures.

The diagnosis not being possible, the morbid process continues, and the abscess may ulcerate, the pus finding its way into a neighboring bronchus and is expectorated, or ruptures may take into the pleura and set up an empyema. The abscess may become encysted, undergo calcification or caseous degeneration, and the seat of tubercular infiltration. Finally adhesions may form and the pus discharge externally through the integument or into an adjoining viscus. In not a few cases the abscess grows larger and larger until the entire lung is destroyed, giving us the physical signs of pyo-pneumothorax.

Unless the abscess ruptures into the bronchial tubes, and the contents are expectorated, the physical signs are not characteristic of the lesion. Before perforation occurs, we have the evidence of local infiltration and consolidation. The sudden, more or less copious expectoration of pus without marked offensive odor in the course of a pneumonia is characteristic of the rupture of a pulmonary abscess into the bronchi. The physical signs now reveal the existence of a cavity of a greater or less size.

Microscopically we find in the expectoration the *débris* of lung structures, elastic lung tissue. According to Leyden we find in addition fatty crystals, pigment *débris*, hæmatoidin and fibrabun, and finally micrococci, differing from those of gangrene in possessing but ltttle motion and in not giving the iodine reaction.

Differential diagnosis.—It is often difficult to differentiate between abscess of the lung and gangrene, except that in the latter the pathological condition and course may have a causative influence in the production of the results. (Meigs.)

Gangrene and abscess observe a close parallelism; that which determines the results is still somewhat obscure, it is probably dependent upon the infecting agent, upon some radical difference in the pathogenic micro-organisms. In gangrene the constitutional disturbances are profound: the odor is fœtid, pulmonary and elastic tissue not present, and the expectoration is dirty green, fœtid and penetrating.

Localized empyema may be excluded by the absence of a history of previous attacks of pleurisy. The absence of earlier attacks of pleurisies in infants makes the occurrence of encysted empyema rare at this age. In older children, who may have suffered from former pleurisies, adhesions may have formed, and the fluid may be encysted.

Empyema discharging through the bronchi may be excluded by remembering that the amount of pus discharged is large, rupture takes place at a later date, and the physical signs reveal fluid in the pleural cavity.

In hepatic abscess discharging through the bronchi, the discharge is copious, dirty brown and paroxysmal; microscope shows *débris* of live tissue. Peripleuritic abscesses are of rare occurrence and usually secondary to empyema or caries of ribs. In doubtful cases, the examination of the sputum for tubercle bacilli will enable us to exclude tuberculosis.

The prognosis is grave if the complication arises in a feeble subject, or if a typhoid state exist. The dangers incidental to abscesses situated internally, and not amenable to surgical interference must not be lost sight of. In addition to exhaustion and sepsis, hæmorrhage from ulceration of large blood vessels may complicate the case.

Treatment consists mainly in the judicious use of stimulants, tonics, and antiseptic inhalations. The ex-

haustion must be counteracted and infection prevented by proper incision and drainage.

In conclusion, the propositions enunciated by Drs. Spillman and Haushalter (*Revue de Méd.*, Aug. 1888) who record nine cases of abscess following pneumonia, seven being successfully treated by operation, are presented. The following rules are laid down by them for our guidance:

1. Before operation the position of the abscess must be determined as accurately as possible, and especially by puncture.

2. As a rule, a pulmonary abscess should not be opened during the course of an acute pneumonia.

3. Before the abscess is opened it is well to excite pleuritic adhesions, if these do not already exist. This should be done by resection of a rib and suture of the two pleural surfaces, or by means of the actual cautery. According to Rüneberg, however, the absence of adhesions does not contra-indicate operation.

4. The abscess is best reached by resecting a rib and piercing the lung with a Thomas cautery.

5. Free drainage is essential; antiseptic injections are best avoided, dry dressings being preferable.

THE ÆTIOLOGY OF STOMATITIS APHTHOSA.

BY F. FORCHHEIMER, M.D.,

Cincinnati, O.

THE term aphthosa, as old as Hippocrates, has caused more confusion than any other in the nomenclature of diseases of the mouth. Even at the present day, so much or so little may be included in the term aphtha that, for the sake of precision in diagnosis, the word should either be dropped entirely or its meaning be so clearly defined that there can be no reasonable room left for error. Without going into an historical development of this part of the subject, which I have done elsewhere, it will be well to state here that the aphthous form of stomatitis

referred to is that disease first defined by Bohn. To Bohn, at least, belongs the credit of first having published the views of other authors besides himself, which finally culminated in making a precise clinical picture of stomatitis aphthosa. If we could adhere to this, and it seems both feasible and practicable, we would have a definite term for a clearly defined clinical picture. If, on the other hand, any ulceration of the mouth, any small loss of substance in the mucous membrane is called an aphtha —as has been done and is being done—we will never arrive at any exact conclusions. Stomatitis aphthosa is a disease characterized by general and local disturbance: the general disturbance in the form of fever and other constitutional symptoms, the local disturbance as an eruption of aphthæ. The aphthæ (and I differ somewhat from Bohn here though not essentially) are to be looked upon as vesicles modified by the locality in which they appear, *i.e.*, the mouth. They are also changed by the action of moisture, being constantly bathed in saliva from the irritated mouth. When, as is not infrequently the case, the eruption also appears upon the skin around the mouth, there can be no doubt of the nature of the eruption, and if, as I have shown latterly in one case, applications of warm water are kept up constantly, the course of these vesicles will be seen to be the same as of those within the mouth. In brief, then, we would call stomatitis aphthosa a vesicular eruption in the mouth with certain constitutional symptoms.

The cause or the causes of this disease have eluded detection; every one who has written on the subject has his own views to a greater or less extent; without disregarding the views of others, there is always one special pet view on this subject. Perhaps the greatest number of authors is agreed on ascribing this disease—like all others —to those glittering generalities that are so well known to all of us in connection with the ætiology of children's diseases. The one calls it struma, the other scrofula, the third malnutrition, the fourth tuberculosis, and so on *ad infinitum*. I am not prepared to say that unhealthy

children are more predisposed to this disease than healthy ones. Whatever our theoretical views may be regarding the vulnerability of children otherwise affected, my observation has led me to believe that the general condition of a child, as far as we can see, has nothing to do with an attack of stomatitis aphthosa. That there is an individual predisposition I firmly believe; in what this consists I do not know; children in the best of health will have an attack sometimes without an apparent cause. Teething, as everywhere else, has been accused of producing this trouble. It can hardly be out of place to say that this view is simply based on the method of reasoning: *post hoc ergo propter hoc.* If we were to accept everything that follows or accompanies the coming through of teeth as being produced by teeth, we would better close the book as far as the ætiology of disease for the first three years of life is concerned. It can be proven beyond a doubt that aphthæ are not tubercular, therefore, not scrofulous nor strumous. There is a rare form of tubercular stomatitis, so rare that every case ought to be recorded, but, as far as I am able to learn, it has nothing in common with the trouble we are now discussing.

It is quite positive that no evidences of tuberculosis have been found in all the bacteriological investigations made by others as well as by myself. Catching cold has, as always, been brought to bear its burden in this disease; so little that is positive can be said in this direction that the less we say the better.

After these general causes are disposed of, the most common belief will probably be found to be that aphthæ are always due to a deranged stomach. This is a very natural view for aphthæ are, as a rule, associated with some derangement of the alimentary canal. The routine practice will be about as follows: ulcers in the mouth a positive indication for rhubarb, calomel or hydrargyrum cum creta. A careful examination of each individual case will prove that the child was ill before the sore mouth came on, that, as it were, the gastro-intestinal symptoms represent a sort of period of invasion for the disease. No

one would for a moment believe that the eruption of scarlatina comes from a deranged stomach although it, of all diseases, is most frequently preceded by vomiting. Errors of diet are so common in childhood that it is almost impossible to exclude them in reckoning upon causes for disease. I have seen three children in the same family have an attack of aphthose stomatitis; in the two older ones error of diet could be taken into consideration, in the third this was impossible as he had taken nothing but sterilized milk.

Yet we will constantly be confronted with the statement that the child's stomach has been deranged for the last few days, now the ulcers have appeared and certainly there must be some connection between the two. It is almost impossible to conceive of the mucous membrane of the mouth being affected by continuity from that of the stomach; the nervous connection is a very remote one, but the clinical fact remains as above stated. If the alimentary canal is primarily the cause of the trouble there is but one explanation, and that is, that a something is excreted by the saliva which causes these ulcers to be formed. But, for various reasons, the mechanism is not so simple for aphthæ; it might be called upon for explanation of the existence of those small follicular ulcers so common with some patients, and would possibly be satisfactory; in aphthous sore mouth there is another factor that seems absolutely essential.

For the last ten years the view has been gaining ground that stomatitis aphthosa is the manifestation of the foot-and-mouth disease in cattle. This view was first brought forward by Sagar in 1764, and has been corroborated by many observers in various parts of Europe (see Bollinger, Ziemssen, Vol. III., p. 581). Hertwig (1834) was the first to prove the connection by direct experimentation; he and three other physicians drank infected milk, and all three were attacked by stomatitis aphthosa; in Hertwig's case the eruption also appeared upon the hands and feet. These results were verified by Jacob in 1846.

On the other hand, a great many authors, and Cayrim, lately, have had occasion to give their evidence on the other side of the question. It must be confessed that the number of observers which has been able to verify this connection is steadily increasing and that the view itself is being more generally accepted. This would place the disease among the class of infectious diseases. Admitting that this view is correct for Europe, according to my information from veterinary surgeons and their literature it could not be true for this country.

Foot-and-mouth disease is an exceedingly rare disease in this country—it is claimed by some that no well-authenticated case is on record.

From the report of the United States Treasury Cattle Commission, 1883 (*American Veterinary Review*, vol. vii.), I learn that an invasion of foot-and-mouth disease swept over northern New York and New England in 1871. In 1883 diseased cattle were landed in New York by two ships (France and Nessmore); these ships are employed to carry cattle, and the cargo which was returned to the other side became infected on board, as the period of incubation seems to be only thirty-six hours. This and other previous experiences (see a long correspondence, Report Department of Agriculture, Diseases of Animals, Washington, 1881) give rise to the idea, held in Europe, that the foot-and-mouth disease may originate in this country. In 1884 Dr. A. A. Holcombe (*Veterinary Review*, vol. viii.) reports an epidemic in Kansas, but ill effects in human beings are not mentioned. In the same year George H. Bailey, D.V.S., reports an epidemic in Portland, Maine, brought in by Hereford cattle. These are the only records of epidemics in this country I have been able to find; it is claimed by veterinary surgeons that no case has ever originated in this country, and by some, that no well-authenticated case is on record, as has been stated before.

However this may be, I have taken the trouble in every instance to look into the milk supply, and have questioned the surgeon in attendance upon the dairies from

which the milk supply has been obtained, always receiving the answer that there never had been a case of foot-and-mouth disease in Cincinnati. Infection would be the most rational explanation for the causation of the disease, and in order to determine whether there could be a *contagium vivum*, I have had the aphthæ examined bacteriologically, all precautions being observed and all the various methods being employed. The result, where positive, was to show the presence of pus-producers, and nothing more.

Dr. Eugene Fränkel (Virch. Archiv., Bd. 113, 111) describes the process as a pseudo-diphtheritic one (Weigert), practically agreeing with Baginsky (1884), who found micro-organisms, cocci, and a few bacilli. Fränkel examined three cases bacteriologically, in two of which he found the staphylococcus pyogenes citreus (Passet), and in the other staphylococcus p. flavus (Rosenbach)—he hastens to the conclusion that these organisms are the cause of the disease, although he has never succeeded in producing any lesions by inoculation in lower animals, thus disregarding one of the most important evidences in regard to the pathogenic nature of an organism.

Accepting the view of the connection between foot-and-mouth disease and stomatitis aphthosa, rejecting the connection for this country, we are left the choice of two conclusions: either we do not have stomatitis aphthosa, or the disease may be produced by more than one cause.

In regard to the first assumption, the non-existence of the disease in this country, I believe everyone will agree that we see as well-defined cases here as can be seen anywhere in Europe. I am sure that I have seen as well-marked aphthæ in my own clinic as I have abroad. The variety of the low forms of life found shows, either, that we have not found the pathogenic form, or, that their invasion is a secondary one, due to an invasion upon a soil made fertile by the aphthous process. In the foot-and-mouth disease any abraded surface may become affected, so that both a local as well as a general infection must be accepted ; whether both causes be the same, cannot

be proven at present. Cases are on record, in the human being, in which, upon the drinking of infected milk, the eruption also appeared upon the hands (Hertwig). In these cases the eruption must certainly have been due to general and not local infection. In the absence of pathogenic forms, we must look for some chemical agent that can be carried into the circulation producing an eruption upon the mucous membrane as well as the skin. Purely theoretical as this may seem, it will be found to have been accepted by dermatologists for many skin diseases (eczema, herpes) and only awaits conclusive proof in the isolation of these bodies, which will become possible with the further advances in animal chemistry.

Now, taking for granted that a chemical body, formed in the foot-and-mouth disease in cows, carried by the milk, produces stomatitis aphthosa in the human being, that we have stomatitis aphthosa in the absence of this foot-and-mouth disease, the inevitable conclusion must be that more than one cause can be found for this disease. Possibly, when bacteriological methods will become still more developed, a lower form of life may be found as the first cause of this disease; for the present the view seems untenable, especially as it has been proven that inflammation may be caused by other agents than lower forms of life. (Grawitz, Bucher, and others.)

In addition, we must be reminded of the fact that aphthæ are frequently associated with other diseases: pneumonia, intermittent, gastro-intestinal disturbances, the acute exanthemata, etc., so that the position of multiple causes would become very much fortified. It seems out of the question to accuse a local cause, although wounds and burns may produce a lesion that cannot be distinguished from aphthæ, so that, in this direction, there seems nothing left but the acceptance of some general causes indicated before. The question would arise: how do these causes produce lesions in the mouth ?

It has always seemed to me that aphthæ were nothing more nor less than the eruption of herpes. The reasons for this are as follows: If we make an artificial

aphthæ (Gerhardt, Bohn, and myself) by any cause which would produce a vesicular eruption upon the skin, the lesion is identical with the one we find in stomatitis aphthosa; in a great many cases of stomatitis aphthosa we find herpes facialis, the eruption being continued from the mucous membrane to the skin, and lastly, the diseases in which herpes occurs most frequently are those in which we commonly find that form of mouth trouble. If we take a vesicle and develop it in the mouth, it will, as has been stated before, undergo the same changes as an aphtha. In brief then, the ætiology of stomatitis aphthosa, in the great majority of instances, is to be studied as the ætiology of herpes non parasitica. Dermatologists regard herpes, especially herpes zoster, as a disease of nervous origin (Bärensprung, Kaposi, Sattler, and others); this would include the herpes secondary to other diseases, for no one can claim for the herpes facialis of pneumonia that it is a zoster, occurring, as it usually does, upon both sides of the face in children (Thomas). The same can be said for stomatitis aphthosa; it does not follow the course of any one nerve, exactly, although in the majority of instances the eruption will be found along some branch of the fifth pair of nerves, especially the lingual.

Verneuil (Annales de Dermatologie, 1885) states that herpes may be of traumatic origin; when a nerve is injured the eruption will appear at its peripheral distribution.

In those cases, then, in which we have been able to find the pathological lesion of herpes, it has been found to be some irritative change in the ganglion from which the nerve arises, or in the nerves themselves. Naturally there must be a something which produces this lesion and it would not be an unreasonable supposition to look for this something in the form of a body the result of bacterial activity or some faulty process in the digestive apparatus. A similar process, not exactly analogous, will be found in diphtheria, where the Loeffler bacillus produces the local manifestations and the toxin the remote ones. If we take the poison of the foot-and-mouth disease, this will represent the toxin of diphtheria, already

formed, which, absorbed from the intestinal tract, produces the general disturbances and finally the aphthæ, as the result of irritative changes in the nerve ganglia. This something, called ptomaïnes by the modern French writers, does not require for its origin the presence of microbes. It may have its orgin within the body, as the result of a perverse digestive process, within the alimentary canal, the liver, or even during the process of reduction within the circulation.

Parrot and Erb were the first to have claimed that herpes might be looked upon as an infectious process, and Hardy (1885), Delétang (1886), Landouzy (1883), and Feulard (1888, Dictionnaire Encyclopedique des Sciences Médicales) have continued the theory, Feulard arriving at the same conclusions that I have expressed above.

Nothing more can be claimed for this view than that it is an hypothesis, with many things necessary to be proven before it can become susceptible of positive proof. The original poison must first be isolated, it will then have to be detected in the circulation, and finally, it will have to be proven that this poison is capable of producing aphthæ. But if we grant the connection with foot-and-mouth disease, and at present this is the result of observation rather than of modern scientific reasoning, it would seem that there is justification for the view expressed before. Although so much is left to be proven before this view can be accepted, we are nevertheless justified in expressing it though purely the result of observations in the various forms of herpes, and deductions from these observations.

The last ætiological factor is contagiousness. Aphthæ will be found in several members of one family at the same time. I have had occasion to observe this in several instances. This is the only evidence that could be brought forward to prove the contagious nature of the process. It would seem more natural, however, to claim that the same cause has been operating upon these various members of the same family, although the aph-

thæ will not appear on the same day in each individual. The latter fact could be readily explained by taking into consideration the varied susceptibility of individuals, and the quantity or quality of the poison introduced into each one of these individuals.

In conclusion I would sum up the results, as follows:

I. Stomatitis aphthosa is a disease produced by some form of deleterious material in the circulation.

II. This material may have its origin in various processes, bacterial or otherwise.

III. This material may, therefore, be of various kinds.

IV. This material acts upon a nerve or nerves, or upon a nerve-centre or nerve-centres.

V. Producing an herpetic eruption which is the aphthous process.

DISCUSSION.

Dr. KOPLIK.—I think all of us have had hundreds of cases almost monthly of aphthæ of all sorts, and nobody can appreciate the difficulty of research as we can. We must consider how difficult it is to find out the cause especially as Müller has shown how many different bacteria there are in the mouth. Dr. Forchheimer has done something to bring him into line with the most advanced investigators of the day. He has ruled out those aphthæ or losses of tissue which Epstein has tried to prove were nothing but a result of traumatism and subsequent infection. I allude to the so-called Bednar's aphthæ. In these cases the aphthæ are situated at the posterior part of the hard palate where normally we see a white reflex appearance of the mucous membrane. These cases Epstein has persisted in saying were purely traumatic. He has maintained principally that the nurse rubs most prominently that part of the hard palate wherever the reflex is seen, where the blood vessels are not so numerous as in the other portions of the hard palate and necrosis takes place more readily. He had therefore instituted a rule that no one in his ward was allowed to wash a healthy baby's mouth. After this rule was adopted where cases of these so-called aphthæ were found in the wards of certain midwives, it was considered that this rule had been transgressed. I expect to see the time when Professor Epstein's views will be vindicated and these cases excluded from the infectious forms of stomatitis.

DR. PUTNAM.—I want to ask Dr. Forchheimer whether it is possible to consider this buccal herpes as practically the same thing as herpes zoster and whether herpes zoster has been shown to have a bacterial origin. He excludes reflex disturbances of digestive organs, and yet I think this disease might be due to some reflex causes. Of course, both may some time in the future be traced to bacteria, but if herpes zoster may be due to irritation of nerves, why not herpes labialis and aphthæ. I am very glad this subject has been brought up for in this country the word "aphthæ" has no particular meaning. I have seen follicular tonsillitis called aphthæ.

Dr. ROTCH.—These terms have been used very loosely and I think we, as a society, should take a decided stand and possibly drop the word altogether, as Dr. Forchheimer has suggested. It would tend to greater exactness. I never use the word except to say to my students that it has been used very indiscriminately. I am accustomed to use the word stomatitis with an adjective, such as ulcerative to differentiate the form. Dr. Forchheimer has done so much good work on this subject that he has become an authority in it. Now if he would suggest how to do away with this term that means nothing, we shall feel greatly indebted to him.

Dr. ADAMS.—It is a singular thing that occurring in the same family in two or three children there is no contagiousness. I have seen five or six children in a wealthy family all taken sick at the same time with the disease, in this city. It is on this ground that we have to fight the homœopaths most frequently. During an attack of diphtheria they call all these cases diphtheritic, and of course great glory redounds to them in curing them so promptly. The sanitary inspector in Georgetown has been called to houses where he had examined the children and where there was no diphtheria but a stomatitis or tonsillitis. We must separate the catarrhal from the ulcerative form because it is a distinct lesion occurring in the buccal mucous membrane and on the outside of the lips in some children. It occurs in the same family and in members occupying the same house, thus appearing to be contagious. I think Dr. Forchheimer agrees with me in that I do not believe that dentition ever caused any disease of this nature, although it occurs about the period of dentition.

DR. ROTCH.—I do not admit that dentition has anything to do with the cause. To return to my former suggestion, can we not have a general term in place of the

word aphthous, can we not, for instance, call it infectious stomatitis, until we get a proper term for the disease? This is what I have been accustomed to do in my endeavors to make the nomenclature of diseases of the mouth more clearly understood by students.

Dr. PUTNAM.—I think this is an excellent idea of Dr. Rotch's, and I move, subject to suggestion, that Dr. Forchheimer be appointed a committee to announce a nomenclature of diseases of the mouth, including such as can be put into definite classes. I think it a good plan to drop this word for no one knows what it means. Perhaps Dr. Forchheimer would like some one associated with him, and I would suggest that Dr. Rotch serve with Dr. Forchheimer on this committee. A motion was made and carried appointing Dr. Forchheimer and Dr. Rotch a committee to report on this subject at the next meeting.

Dr. FORCHHEIMER.—As to the suggestion of Dr. Putnam concerning a reflex cause of aphthæ, I would admit that such a thing may occur, as far as the investigations have gone, however, the reflex causes may be excluded. Kaposi and others have looked into the subject and find that there is something more than a change in the trophic ganglion of the chords, or if it is the facial nerve, a change in the ganglia there. I was astonished to find that in 1883 another party had worked out the thing as I had. I think there is some connection between digestion and aphthæ. There is something formed that passes through the liver undisturbed, passes into the circulatory system and affects the nervous system, thus indirectly. These products may be formed in the change from albumen to albumoses, or may be produced in the tissues themselves. What astonished me in working up the literature was, that the Frenchmen had worked out these things before the Germans. As to dropping out the word aphthæ, in the series of articles which will appear in book form, I have adhered to the word aphthosa. I have retained the term stomatitis and used the word aphthosa as an adjective, out of respect to Bonn. The history of the word goes back into the writing of Hippocrates. From him we have retained the term and many different things have been called aphthæ. If we understand what aphthosa means, there can be no difficulty in retaining the term, and we place ourselves in direct connection with all the nations, German, French, and others that use it. The English have a form they call follicular. The Germans use the term aphthosus. Aphthæ can be dropped, but I think

aphthosus should be retained. As to the disseminative forms, it has occurred to me that they are badly developed cases of stomatitis aphthosus. As to Dr. Adams' remarks stomatitis diphtheritica, is extremely rare. I know many practitioners call anything occurring in the mouth diphtheria, including stomatitis aphthosa, which is not so. The application of remedies to the fauces the use of sprays, the use of very hot applications, all these may produce lesions in the mouth. But in addition to this stomatitis is an uncommon complication of diphtheria, and these forms rarely become diphtheritic. When an eruption occurs in the mouth in diphtheria it is usually stomatitis aphthosa, but it rarely becomes diphtheritic. I do not believe that the disease is contagious. The form that is the result of foot-and-mouth disease is not contagious. Three persons drinking infected milk will, as a rule, get stomatitis aphthosa, but those associating with them will not. The only possible way in which I can explain the occurrence of several cases in the same family is from the operation of the same causes; the children all take the same food and the same causes apply to and affect all. As to Dr. Koplik's remarks, Epstein's first work is more in the direction of ascribing the trouble to some anatomical condition, which predisposes these parts on account of attrition simultaneously with Epstein; I reached the same conclusion—that these forms were due to mechanical irritation. They occur only where traumatism can occur. Bednar's aphthæ occur very rarely in this country. We sometimes see a similar form upon the hard palate, the result of badly constructed nipples, causing a large, flat ulcer. The pathology of this form which seems to be clearest is the same as Bednar's.

Dr. ROTCH.—Whether we change these terms or not it seems to me that the committee will be useful just the same, and we shall keep in accord with the leading minds of Europe.

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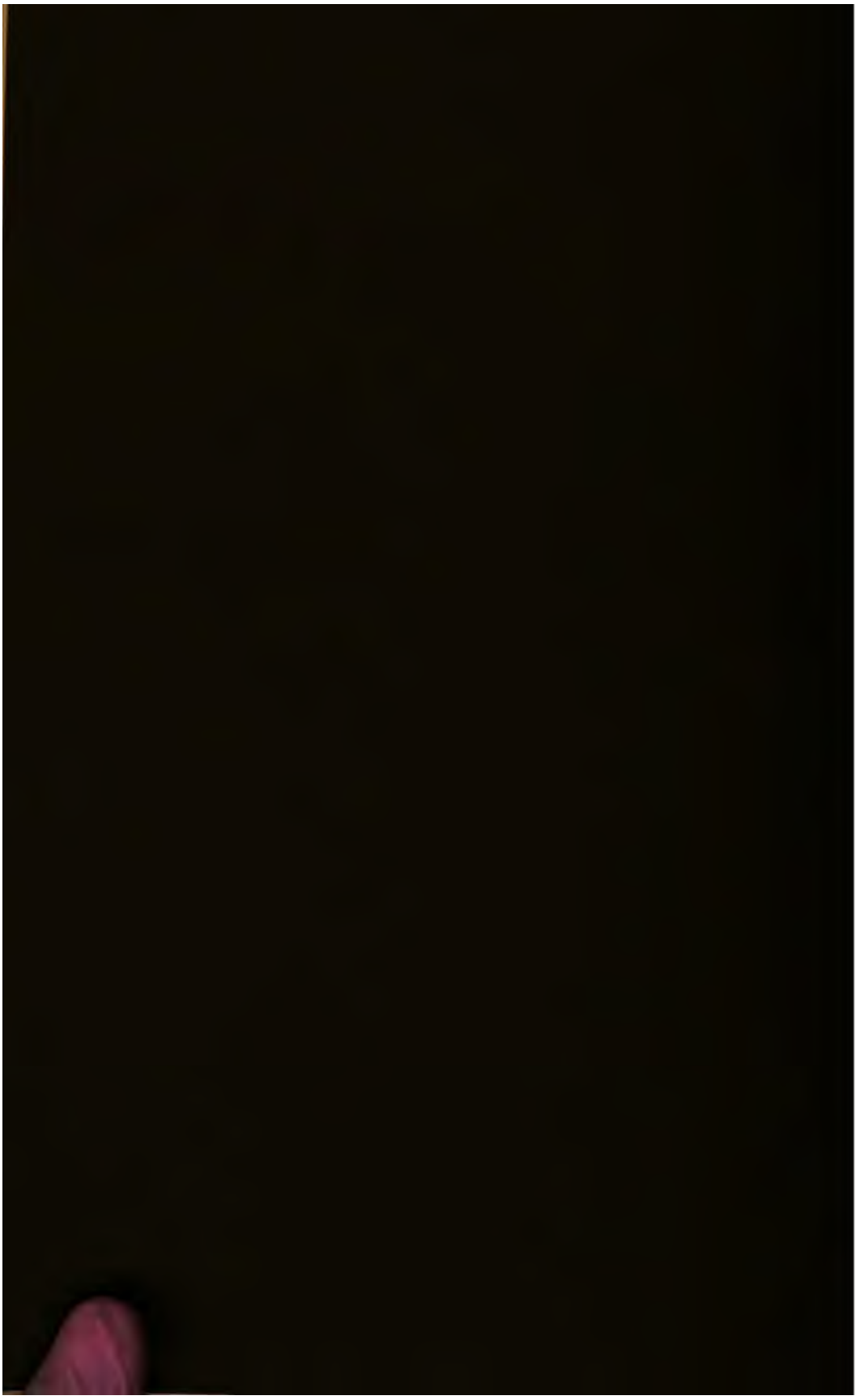
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